"THE STUDY OF PATTERN OF INTRACRANIAL HAEMORHAGES IN FATAL HEAD INJURY CASES OF ROAD TRAFFIC ACCIDENT"

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BONAFIDE CERTIFICATE

This is to certify that the work embodied in this dissertation entitled **"THE STUDY OF PATTERN OF INTRACRANIAL HAEMORHAGES IN FATAL HEAD INJURY CASES OF ROAD TRAFFIC ACCIDENT"** has been carried out by **Dr. A.NIRMALA,** Post Graduate student under my supervision and guidance for her study leading to Branch XV M. D. Degree in Forensic Medicine during the period of May-2015 to May-2018.

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DECLARATION

I **Dr. A.NIRMALA**, solemnly declare that this dissertation entitled **"THE STUDY OF PATTERN OF INTRACRANIAL HAEMORHAGES IN FATAL HEAD INJURY CASES OF ROAD TRAFFIC ACCIDENT"** is the bonafide work done by me under the expert guidance and supervision of **Prof.Dr.P.Parasakthi, M.D.,** Professor and Director, Institute of Forensic Medicine, Madras Medical College, Chennai-3.This dissertation is submitted to the Tamil Nadu Dr.M.G.R Medical University towards partial fulfilment of requirement for the award of M.D., Degree (Branch XV) in Forensic Medicine.

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DEDICATION

To My Daughter Miss.B.Aishwariya, B.A (Political Science) and My Parents for their immense support and affection

ABBREVIATIONS

MCF	-	Middle cranial fossa
ACF	-	Anterior cranial fossa
PCF	-	Posterior cranial fossa
FF	-	Fissure fracture
RTA	-	Road traffic accident
VS	-	Versus
ICH	-	Intra cerebral haemorrhage
EDH	-	Extra Dural Haemorrhage
IVH	-	Intra ventricular haemorrhage
SDH	-	Sub dural haemorrhage
SAH	-	Sub arachnoid haemorrhage
FTP	-	Fronto tempero parietal.
RT	-	Right
LT	-	Left

CONTENT

INTRODUCTION

INTRODUCTION

India is undergoing major economic and demographic transition coupled with increasing urbanization and motorization. Among the top ten causes of mortality in the country, road traffic accidents (RTAs) was the tenth cause before two decades, but with the increasing urban expanse and lifestyle changes, it is projected that road traffic accidents will occupy the fifth position in the list of major killers and second position among the causes of disease burden in 2020.

'Head injury' as defined by the National Advisory of Neurological Diseases and Stroke Council, is a morbid state, resulting from gross or subtle structural changes in the scalp, skull, and/or the contents of the skull, produced by mechanical forces. It is also defined as 'any injury that causes lesion or functional damage of cranium, meninges and brain'. Head injury is the most frequent cause of death in trauma related- deaths.

According to WHO estimates [1], about 1.24 million people die each year as a result of road traffic crashes. Between 20 to 50 million more people suffer non-fatal injuries, with many incurring a disability as a result of their injury. In India, according to Ministry Of Road Transport and Highways (MORTH)[2], during the year 2010, there were around 5 lakhs road accidents, which resulted in deaths of 134,513 people and injured more than 5 lakhs persons in India. It is the leading cause of mortality for young adults of age less than 45 years. Some

of the factors that increase the risk of road traffic accidents in India are unsafe traffic environment, poor road infrastructure and encroachments that restrict safe areas for pedestrians, lack of safety engineering measures, traffic mix, an increasing number of motorized vehicles, unsafe driving behaviour and lack of valid licenses.

Among all the regional injuries, the injury to the head and neck are the most important in forensic practice. The external injury on the head and face may or may not be representative of the extent of the internal injury. A sound practical understanding of the neuro pathological trauma is more essential for forensic pathologist because head injuries provide the major contribution of the death due to assaults, falls and transportation accidents. Road traffic injuries are the leading cause (60%) of traumatic brain injuries followed by falls (20%-25%) and violence (10%).

CT scan is commonly used as the initial diagnostic tool to look for various kinds of lesions in cases of head injury. The aim of CT scanning is to rapidly demonstrate a surgically correctable lesion, fracture of skull and intra-cranial haemorrhages. The gold standard for the post-mortem forensic assessment of neuro-trauma cases is autopsy along with Histopathology examination. Primary advantages of autopsy include critical analysis by meticulous observation with precise dissection and if required taking tissue specimens for further laboratory studies. One limitation of autopsy is that it is subjective and observer-dependent, destroys the tissue morphology and cannot

be used to provide a second opinion in the Court due to the fact that the body tissues cannot be stored for a long time except specimens for histo-pathological examination. Ante-mortem radiological imaging methods such as, CT scan allows an unlimited storage of the imaging data and facilitates second opinions in the course of legal proceedings. Considering that CT scan might play an ever-increasing role in forensic neuro-traumatology

AIMS AND

OBJECTIVES

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1. To find out the magnitude of problem with reference to different host factors.

2. To analyse the significance of nature of injury, part of the brain injured and the time of survival.

3. To study the pattern of intracranial haemorrhages in deaths due to fatal head injuries in RTA.

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY SETTING

A prospective study of fatal head injury cases was undertaken during the period August 2016 to August 2017.These head injury cases were admitted in the Neurosurgery Department of GOVT RAJIV GANDHI GENERAL HOSPITAL, Chennai and succumbed while on treatment.

The autopsy of these fatal head injury cases were conducted at the Mortuary, Department of Forensic Medicine, RAJIV GANDHI GENERAL HOSPITAL Medical College, Chennai.

STUDY POPULATION

The patients who got admitted in the RAJIV GANDHI GENERAL HOSPITAL MEDICAL COLLEGE & HOSPITAL with head trauma due to road traffic accidents and, succumbed during treatment and then subjected to medico-legal autopsy during the above mentioned period of time, were enrolled in this study.

Photographic Evidence of gross features of autopsy were presented for on demand discussion.

INCLUSION CRITERIA

All cases of fatal head injuries due to road traffic accidents subjected for medico – legal autopsy.

EXCLUSION CRITERIA

1. Those patients who died in RTAs with cause of death other than head injury were excluded.
2. Decomposed cases with fatal head injuries, where the interpretation of injuries was not possible due to extensive decomposition were excluded.
3 Coexisting antemortem or postmortem burn injury to head.

SAMPLE SIZE

A convenience sample size 200 cases, who succumbed to head injuries from RTAs were included in the study.

METHODOLOGY

1. A detailed data of socio-demographic profile of each deceased was collected including age, sex, socio-economic status, occupation, religion, marital status and educational qualification.
2. A detailed history pertaining to circumstances, time of accident, day/night, summer/winter/rainy seasons was recorded.
3. Manner of accident like vehicle vs vehicle, vehicle vs pedestrian, direction of vehicles etc. was recorded.
4. Transportation time between the time of accident and arrival to the hospital was recorded.
5. Details of the course of the patient's condition after hospitalization including the details of interventions done or surgery performed were

noted down. All the necessary documents were perused prior to the medico-legal autopsy.

6. A complete autopsy was done and all the findings were recorded in detail. Details were tabulated as per pre-fixed parameters in a standard performa.

Dissection Technique:

The dissection technique of scalp, skull and dura was in accordance with the procedures suggested by Gresham GA and Turner AF and the brain dissection as suggested by Ludwig J [3, 4].

The hair was parted along an imaginary coronal plane connecting one mastoid with the other. The scalp was opened by a transverse incision across the vertex from mastoid to mastoid and flaps were reflected anteriorly up to orbits and posteriorly below the occipital protuberance. The layers of the scalp were examined for extravasation of blood by making multiple transverse incisions over the inner surface. The temporalis muscles were reflected from there attachments on both the sides using a scalpel.

The skull vault was opened using an oscillating Stryker saw (Michigan, USA). The cranium was opened with a 'V' shaped incision starting from a point 3 cm above the supra-orbital ridge in the front, extending to a point just above the ears in the sides and terminating at a point 3 cm above the external occipital protuberance on the back. Sawing was stopped when the oscillating

blade reached the diploe, where the saw meets with little resistance. The skull cap was then separated by gently inserting and twisting the chisel at various places through the cut.

The superior sagittal sinus was opened in the midline by making an incision in the falx cerebri with a scissor and examined for any ante-mortem thrombus. Two parallel incisions and one transverse incision were made over the dura matter on either sides of the falx cerebri resulting in the opening of the dura in four flaps. The anterior part of the falx was cut free from the cribriform plate, and using forceps the falx cerebri was pulled backwards.

The anterior poles of both frontal lobes were mobilized with the left hand, and the exposed cranial nerves and pituitary stalk were cut. The tentorium cerebelli was cut on either side along the petrous ridge of the temporal bone with curved scissors. The remaining cranial nerves were cut and the knife was introduced into the foramen magnum and the spinal cord was cut below the medulla. The brain was delivered from the cranial cavity by giving support with the left hand. With an artery forceps, the dura mater was stripped off from the base and the sides of the skull, which was then examined for evidence of fracture. The pituitary gland was removed and dissected by incising the diaphragm sellae and knocking off the posterior clinoid process with a chisel.

The brain was inspected externally on all the surfaces. The base of the brain was examined carefully for any pathological conditions such as aneurysm, herniations, thrombosis and embolism.

The brain was placed with its convexity upwards with the orbital lobes and occipital poles in a horizontal plane. The brain was dissected obliquely using brain knife from the corpus callosum on the either sides exposing the lateral ventricles, internal capsule and basal ganglia. Then the section was taken in a coronal plane through the mammillary body. The brain was subjected for 1cm thick coronal sections. Each slice was examined before the new slice was made. The brain-stem and cerebellum were sectioned in a horizontal plane.

Finally autopsy findings of fatal head injuries were carried out. Major findings like intracranial hemorrhages such as extradural hemorrhages, subdural hemorrhages, subarachnoid hemorrhages, intra ventricular hemorrhages; brain parenchymal contusions, fracture of skull and lacerations were evaluated in each case.

STATISTICAL ANALYSIS

All the statistical analysis was done using EpiInfo 3.4.3 (2007) software. Descriptive statistics of categorical data were presented as proportions for comparison.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

ANATOMY OF SKULL AND BRAIN[5, 6, 7]:

The Scalp:

Scalp is the soft tissue covering of the skull which extends from the supraorbital ridges anteriorly to the occipital protruberance posteriorly. It is usually described as having five layers:

S: The skin. It contains hair follicles and numerous sabaeceous glands.

C: Connective tissue. A thin layer of fat and fibrous tissue lies beneath the skin.

A: The aponeurosis called epicranial aponeurosis (or galeaaponeurotica) is the next layer. It is a tough layer of dense fibrous tissue which runs from the frontalis muscle anteriorly to the occipitalis posteriorly.

L: The loose areolar connective tissue layer provides an easy plane of separation between the upper three layers and the pericranium. It contains the major blood vessels of the scalp which bleed profusely upon injury, partly due to the absence of venous valves found in the circulation below the neck. Hence is called ‘the danger area’ of the scalp.

P: The pericranium is the periosteum of the skull bones.

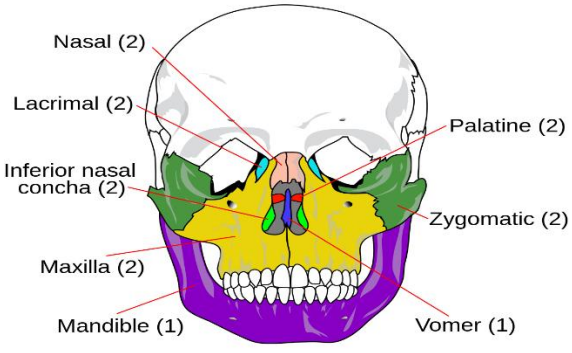
The blood supply of the scalp is via five pairs of arteries, three from the external carotid and two from the internal carotid arteries.

The Face:

The face extends superiorly up to the hair line, inferiorly up to the chin and the base of the mandible; and on each side to the auricle. It is connected to underlying bones by loose connective tissue, in which muscles of facial expression are embedded. The deep fascia is absent in the face. The facial skin is very vascular. The wounds of the face bleed profusely. The facial artery is the chief artery of the face which is a branch of external carotid artery. It is also supplied by a small artery, branch of superficial temporal artery called the transverse facial artery. The veins of the face accompany the arteries and drain into common facial and retro mandibular veins and then into the external jugular vein on each side.

The Skull:

The skeleton of the head is called the skull. It can be divided into 2 main parts, namely the neuro cranium and the splanchno cranium. The neuro cranium or the calvaria is the upper part of the brain which encloses the brain. The splanchno cranium is the facial skeleton which includes the mandible. The skull consists of 22 bones.



14 Facial Bones

The calvaria or the brain case is composed of 8 bones:

Paired: Parietal, Temporal

Unpaired: Frontal, Occipital, Sphenoid, Ethmoid

The facial skeleton is composed of 14 bones:

Paired: Maxilla, Zygomatic, Nasal, Lacrimal, Palatine,

Inferior nasal concha.

Unpaired: Mandible, Vomer

The Meninges:

The membranes covering the brain are called meninges. They are the outer duramater, the middle arachnoid and the inner piamater. The duramater consists of two layers – an outer endosteal layer and an inner meningeal layer. The cranial venous sinuses lie between these two layers. Space between the skull and dura is extradural space. Arachnoid membrane is a thin transparent membrane, loosely surrounds the brain. It does not dip into the sulci, except in the longitudinal fissure and stem of the lateral sulcus. Space between the dura and arachnoid is called subdural space. Piamater is a thin vascular membrane. It dips into the sulci of brain. The fold of pia covering chroid plexus is called as 'telachoroidae'. Space between pia and arachnoid is called subarachnoid space, which contains the cerebrospinal fluid. FalxCerebri is a sickle shaped fold of duramater between two cerebral hemispheres. Tentorium cerebelli is a crescent shaped fold which roofs over posterior cranial fossa.

Cerebrospinal fluid:

It is a modified fluid derived from blood. It is secreted from choroid plexus of lateral ventricles and the 3rd ventricle. It is approximately 150 ml in quantity, secreted at the rate of 200 ml/hour. It is reabsorbed into the capillaries contained in the arachnoid villi. It functions as the lymph of brain and also has a cushioning effect on the brain protecting it from injuries.

Brain:

The major regions of brain are the cerebrum, basal ganglia, diencephalon, brain stem and cerebellum.

Cerebrum: The cerebrum consists of 2 cerebral hemispheres, the right and the left, separated by the longitudinal fissure and falxcerebri. It has 4 lobes on each side namely, frontal, temporal, parietal and occipital lobes. The cerebral hemispheres are divided into the grey matter, which contains the neuronal bodies and the white matter, which contains the axons. The 2 hemispheres are connected to each other by a band of white matter called corpus callosum.

Basal Ganglia: It is constituted by the deep grey matter nuclei namely, caudate, putamen, globus pallidus, and subthalamic nuclei.

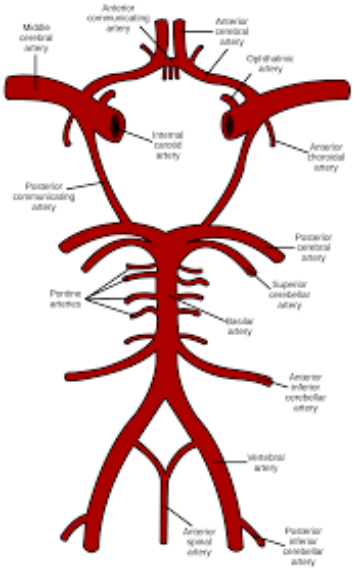
Diencephalon: The diencephalon is located centrally within the forebrain. It consists of the thalamus, hypothalamus and epithalamus, which together enclose the third ventricle.

Brainstem: The brain stem is made up of the upper midbrain, middle pons and lower medulla. They contain all the vital centres controlling the cardiorespiratory functions. The cavity of the midbrain is the aqueduct of sylvius, connecting the 3rdand the 4th ventricles. The 4th ventricles lie between the pons and medulla anteriorly and cerebellum posteriorly.

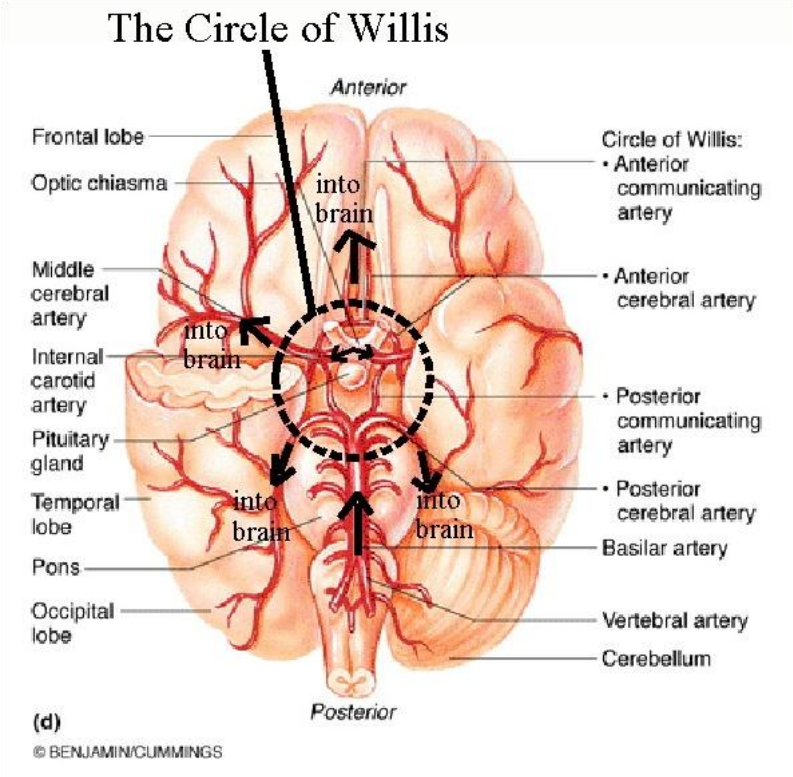
Cerebellum: It is the largest part of hindbrain, situated in the posterior cranial fossa. The two cerebellar hemispheres are connected by a midline vermis.

Blood Supply of the Brain:

Arterial Supply: The right and left internal carotid arteries form the anterior cerebral circulation, each dividing into anterior cerebral and middle cerebral arteries. The posterior cerebral circulation is formed by the right and left posterior cerebral arteries, branches of basilar artery. All the arteries together form the Circle of Willis.



Circle of Willis



Venous drainage:

External cerebral veins draining the superficial regions of the cerebral hemisphere drain into the dural venous sinuses. The thalamo striate and choroidal veins draining the deeper regions drain into the internal cerebral veins, which unite to form the great cerebral vein. The median great cerebral vein drains into the straight sinus. The inferior cerebral veins drain into the cavernous sinus.

Mechanism of head injuries:

Fracture of the skull:

Rowbotham's hypothesis[8]**:**

1. The direct application of force to the skull.

   Local deformation of segment of a skull at the site of impact.

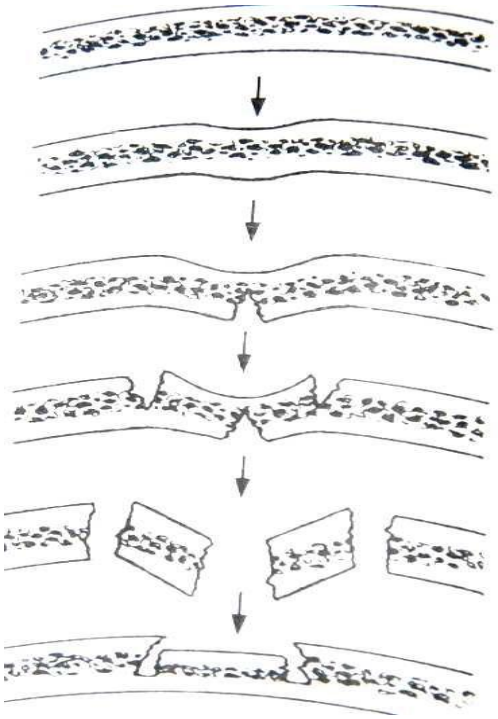
   General deformation of the skull

2. The indirect violence.

The direct application of force to the skull

The bones of the skull have limited elasticity and if the force is applied to vault of the skull, the bone may bend without fracture. If the bending is sufficiently great, then the bone fractures. The sequence of events has been given, showing the local deformation at the site of impact. At the site of impact the bone is indented in the form of shallow cone, the inner table. At the apex of the cone the inner table is stretched and outer table is compressed. At the periphery the convexity is directed outside. If the limits of elasticity are crossed, a fracture confined to inner table will results. With application of greater force both the inner and outer tables of the skull are fractured. The extension of break at the apex through outer table and the outer table at the periphery will results in depressed comminuted fracture.

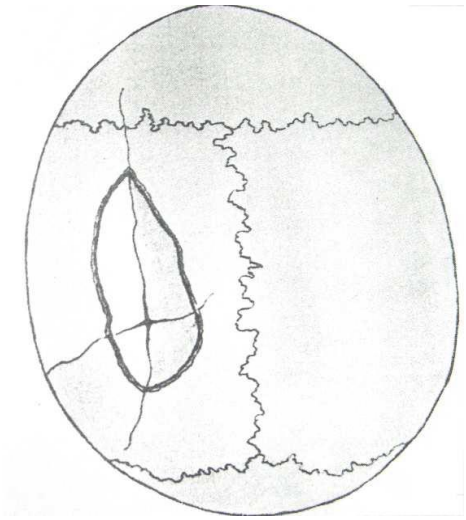
Direct Application of Force[9]



Fractures of the skull due to general deformation:

The skull behaves like anelastic sphere, so that when compressed in one plane it bulges in other direction.

General Deformation[9]



Fracture due to general deformation is usually the fissure fracture and they occur in that part of the skull away from the site of application of the force. The bones often vary in their thickness, enclosed in strong ridges and buttresses of the bone.

Depressed Fracture of Vault of Skull

Fractures of the skull due to indirect violence:

It is mainly due to indirect force applied to face and chin or force applied upwards through the spinal column, like fall from height on buttocks or on feet.

The hypothesis of Gurdijan et al[10]:

The damage done to the bone is due to the way in which the energy is applied, although the traumatizing object and the skull thickness also matters. The magnitude of the force may be important in designing the vehicle and protective headgear and seat gear. The kinetic energy developed under these conditions of impact depends on the formula.

EK=1/2 mv2

Where,

EK - Kinetic energy

m - Mass (in lb)

v - Velocity (feet per second)

The experiment has been conducted which established certain physical facts.

All the impacts produce deformation of varying intensity if the energy expenditure is adequate. Occasionally contre-coup out-bending was observed diagonally opposite the point of impact. The skull bends inwards under the direct point of impact and radiates outside from the point of impact.

The area of in bending can rebound or fail in which case a depressed fracture results. The depressed fracture is decided partly based on the skull and the weapon but mainly by the velocity of injuring object. The tearing apart force in area of out bending may result in linear fracture, which starts at a considerable distance from the point of impact and run towards it. The fracture line general reaches the impact area: but if energy is insufficient, the fracture may remain limited and not reach the impact area. Studies also showed that hair, scalp and intracranial contents did not alter the position of the fractures that resulted. The same amount of energy can produce different types of skull fractures.

Injuries to the scalp:

The scalp is often, though by no means invariably, damaged in trauma that causeinjury to underlying skull and brain. The usual range of abrasion, contusion and laceration may be inflicted, though the modification factor is presence of hair, which may deflect a tangential blow or partly cushions the direct impact. In hair covered areas, care must always be taken at autopsy to palpate the scalp in all cases in which possibility of injury, otherwise abrasion, swelling, bruising and laceration may be missed.

Abrasion:

Brush abrasion is less common than any other place because of the protective effect of the hair which tends to prevent or blur the pattern. Lesser degree of abrasion is inevitably missed.

Bruising:

Marked swelling is the common features of extensive bruising, as the liberated blood cannot extend down wards because of rigidity of the underlying skull. Blood may be present in pericranium (periosteum) that is closely applied to the outer surface. Bleeding underlying the scalp may mobilize especially under gravity resulting in “Black eye.” where the hematoma under the anterior layer of the scalp gravitates to appear in the orbit. The shape of the inflicting weapon or object can be poorly reproduced in the scalp because of padding effect of the hair.

Laceration:

It bleeds profusely and fatal blood loss can occur from extensive scalp injuries. Flaying injury similar to those caused to the lower limb may happen in RTAs. A post mortem injury may bleed profusely when inflicted soon after death. There is no reliable way of resolving it. Blunt laceration of the scalp will have the following features -bruised margin even though zone may be narrowed, head hairs crossing the wound, which may not occur in the cut, facial strands, hair bulbs and small nerves and vessels in the depth of the wound.

Injuries on the posterior aspect of the scalp are commonly caused by falling, especially in inebriant victim. Fall may usually injure occipital

protuberance, forehead and parieto temporal area. Injury on vertex should always raise suspicion of assault.

Fractures of skull:

Linear fractures are straight or curved fractures, often of considerable length. They either radiate out from depressed zone or arise under or at a distance from the impact area from bulging deformation. They may involve inner table or outer table commonly traverses both. A common basal linear fracture is one that passes across the floor of middle cranial fossa often follows petrous temporal or greater wing of sphenoid bone into the pituitary fossa. This frequently continues symmetrically across the other middle cranial fossa separating basal skull in to two halves, usually caused by heavy blow to the side of the head; this lesion is sometimes called as motorcyclist fracture or Hinge fracture. Hinge fracture is divided into three categories-

Type I - runs in the coronal plane, extending from lateral end of one petrous ridge through sella turcica to the contralateral petrous ridge. This type of hinge fracture is the most common type of fracture.

Type II - runs from front to contra lateral back through sella turcica.

Type III - runs from side to side in coronal plane but do not pass through sella turcica.

In children and young adults, a linear fracture may pass into suture line and cause ‘diastasis’ or opening of weaker seam between the bones. This is most often seen in sagittal suture between two parietal bones. Such fractures are called as sutural fracture or diastatic fracture.

Ring Fracture occurs in posterior fossa around foramen magnum and often caused by fall from height on to the feet. If the kinetic energy of the fall is not absorbed by the fractures of the legs, pelvis or spine, the impact is transmitted up the cervical spine.

Fissure fracture of Vault of skull

Mechanism of Brain damage and intracranial haemorrhage.

Holbourn’shypothesis[11, 12]:

The shape of skull and brain is important in determining the localization of injuries when head is subjected to the blow. It has been postulated that brain tissue is injured when the constituent particles are pulled so far apart that they do not join up again properly when the blow is over. The shear strain at any point in the brain is a rough measure of probability of injury at that point. As the brain tissue is incompressible but easily deformed, shear strain are the cause of injury, where as it is not due to compression and rarefaction strains. The forces, which are transmitted from rotating skull to the brain act chiefly upon the surface of the brain. The strain will be maximally developed where the great force is applied to the brain for example at the poles.

Acceleration or deceleration injuries are due to sudden movement of the head, the instance after injury, resultant production of intracranial pressure gradient and subjecting brain to both shearing and tensile forces. An impact force to the head can produce linear acceleration or rotational (angular) acceleration. In linear acceleration, force passes through the center of the head, accelerating head in straight line. In rotational (angular) acceleration, the force does not pass through the center and thus head will rotate about its center. Impact to the front and back of the head tends to produce linear acceleration while those to the side produce a combination of linear and angular acceleration.

The effect of linear acceleration or deceleration on brain tissue are demonstrated in Holbourn's model. It demonstrated the fallacy that the brain lies loose in the skull and when head is struck it rattles about like a die in a box,

thereby causing coup and contre-coup injuries. Rotational shear strain causes considerable damage.

The pathology of brain injuries:

There are three types of primary damage that can occur at the time of head injury namely diffuse neuronal injury, contusion and laceration. Any one or combination of any lesion can occur. Oedema and massive intracerebral haemorrhage are secondary phenomena, even though they may develop soon after the injury.

Diffuse neuronal injury:

In some cases of head injuries, there are no naked eye lesions in the brain tissue. Routine histological examination reveals certain microscopic hemorrhages in the tissues. Rowbotham suggested that it probably depends on widespread intracellular disturbance and damage to synaptic junction between neurons.

Diffuse neuronal injury may be wholly or partially reversible. Death may be due to added injuries but if the gross lesion is limited in its extent then it is probable that diffuse neuronal injury is the primary cause of death. It is impossible in that case to establish cause of death based on only autopsy findings; if clinical information is available the diagnosis depends on clinical information. Whether there was some disturbance in consciousness which is followed by progressive failure of central nervous system has to be determined.

Diffuse axonal injury:

In diffuse axonal injury, immediate prolonged coma unaccompanied by an intracranial mass lesion occurs in almost half of patients with severe head injuries. It is caused by sudden acceleration or deceleration of the head. It will vary from mild brain injury and dysfunction to severe irreversible dysfunction and injury, and even death. As the physical force increases, there will be progressive increase in the extent of physiological injury to the axons plus an increase in the amount of immediate structural disruption of axons with immediate cession of all the activities. Diffuse axonal injury depends on acceleration and also the time over which it is acting, hence the diffuse axonal injury is common in vehicular accident, where the time of impact is prolonged due to absorbing material, but rare in fall where there is no absorbing material. It is extremely uncommon for DAI to occur in the fall of a person from same height.

Cerebral Contusion:

Cerebral contusion is a circumscribed area of brain tissue destruction, which is accompanied with extravasation of blood into the affected tissue. Contusions are often multiple and vary in size. Contusions often occur in cortex of the brain and also in deeper tissues. In cortex they are often covered by narrow zone of intact tissue. They are frequently wedge shaped and surrounded by petechial haemorrhages. They may be produced by distortion of

skull or rotational movement of brain in relation to skull. Contusions are not seen in infants rather one can see laceration involving white matter.

There are six types of contusions.

1. Coup contusions occur at the site of impact and are due to in bending bone snapping back, inflicting tensile force injury to brain.
2. Countercoup contusions occur directly opposite to point of impact, they are classically seen in cases of fall from height.
3. Fracture contusions are associated with fracture of the skull.
4. Intermediary coup contusions are hemorrhagic contusion in the deep structures of the brain example in white matter, basal ganglia of brain and corpus callosum.
5. Gliding contusions are focal haemorrhages located in cortex and underlying white matter of dorsal surface of cerebral hemisphere. They are independent of site and direction of impact.
6. Herniation contusions caused by impaction of medial portion of temporal lobes against edge of tentorium or the cerebellar tonsils against foramen magnum.

Cerebral Laceration:

Cerebral contusion and cerebral laceration are fundamentally similar types of disruptive injuries. Laceration is a larger lesion than a contusion. Laceration is usually surrounded by groups of contusions. Laceration is caused by same mechanism as contusion. Laceration is particularly prominent in the

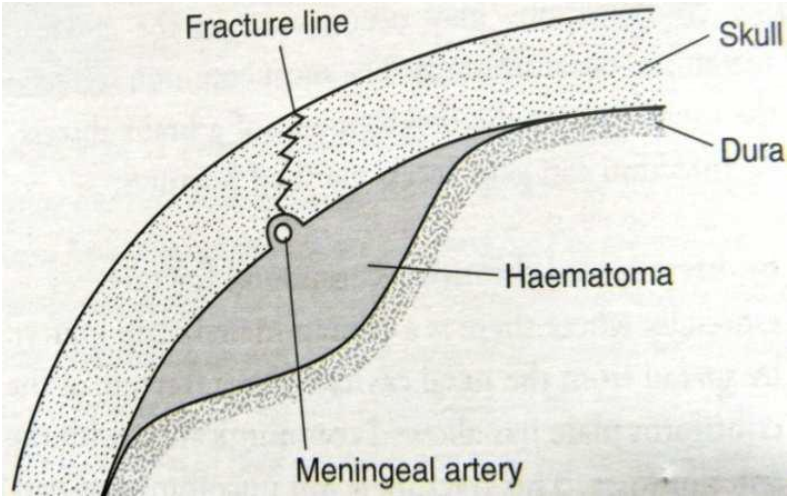
region where the brain is in contact with projecting buttresses and ridges on the inner surface of the skull. Extensive lacerations may be caused by penetration of brain tissue by the fragment of fractured bone. Due to rupture of overlying piamater, laceration is often accompanied by extensive subarachnoid haemorrhage. The healing of the laceration results in development of adhesion, which gives rise to secondary epilepsy a condition which can manifest months and years after head injuries.

When deep laceration involves the ventricles, healing may results in the formation of large glial cysts filled with cerebrospinal fluid. These cysts are known as traumatic porencephalic cysts and must be distinguished from cysts, which develop in zones of contusions and hemorrhages. The walls of the cysts usually show well-marked blood pigment staining.

Extradural or Epidural haemorrhage/ hematoma:

Bleeding between the skull and the dura mater is the least common of the three brain haemorrhages.

Extradural Hematoma[9]



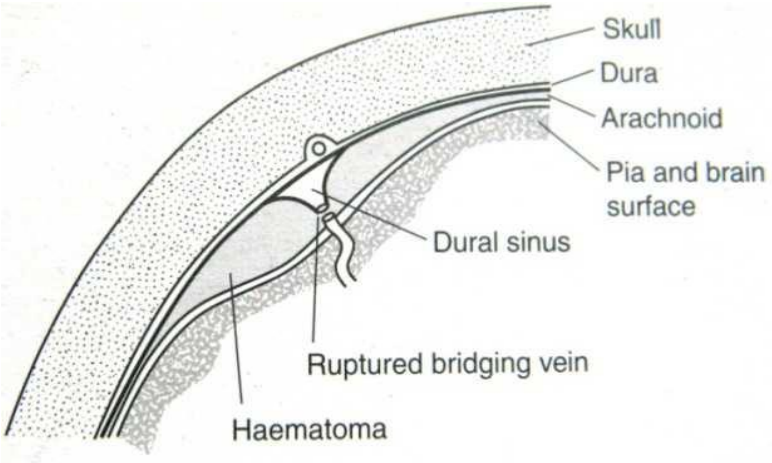
As the duramater is tightly applied to the base of the skull except in the posterior fossa, extradural haemorrhage does not occur in the skull base. In the vault there is a potential space between dura and the bone, which can be separated by the arterial bleed and less often venous bleed. Most extradural haemorrhages are associated with skull fractures. The usual site is parieto temporal area, caused by rupture of middle meningeal artery where they are traversed by fracture of skull. The posterior branch of this vessel is more common than the anterior branch to rupture**.** The vessel usually lies in deep osseous groove in first part of its course. Leakage of high-pressure arterial blood strips back the underlying dura with progressive accumulation of a hematoma. When bleeding is venous, the hematoma rarely reaches a large size as pressure is insufficient to tear back much dura. Extradural hematomas are usually primary impact injuries. They are relatively infrequent and most commonly seen in cases of falls and RTAs. They are infrequent in elderly and

the very young due to strong adherence of the dura to the skull in both these age groups. Chronic extradural hematoma is rare, it is said as chronic when more than 48 to 72 hours has passed from the time of sustaining injury.

Extra dural haemorrhage

Subdural haemorrhage:

Subdural haemorrhage is less often associated with fracture of skull



Subdural Haemorrhage

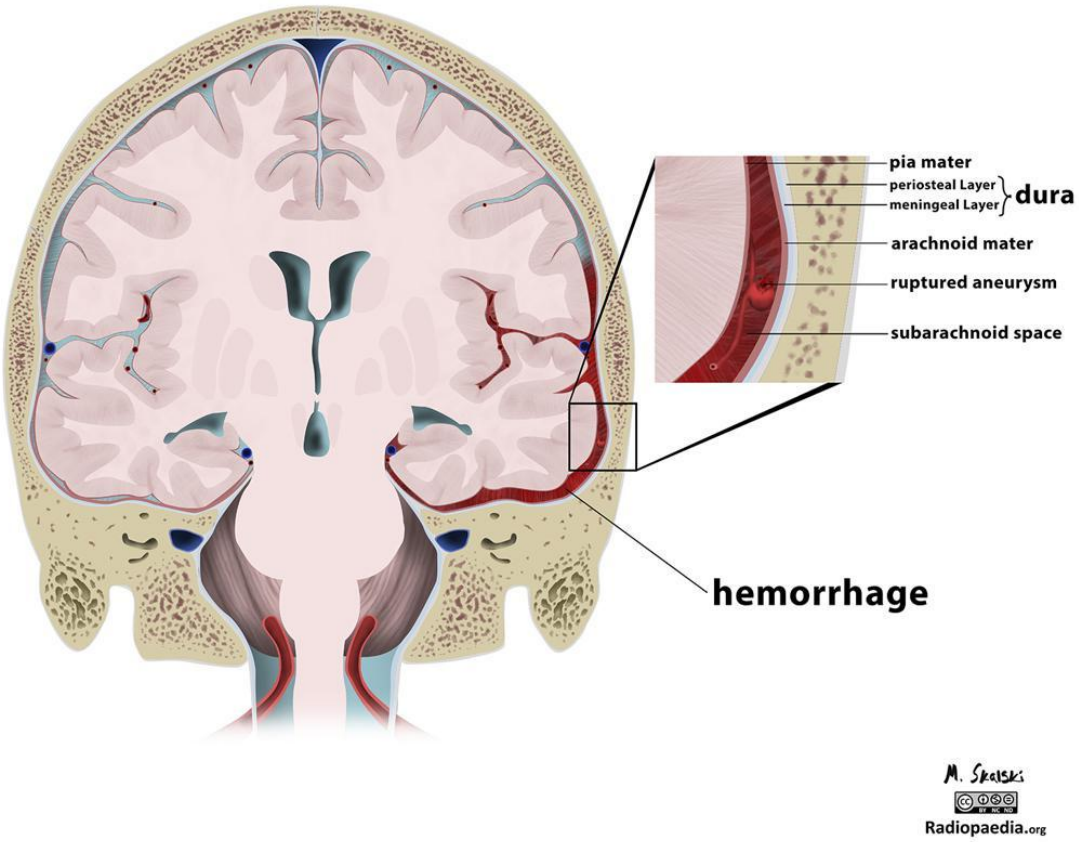
The lesion is traditionally classified into acute, subacute and chronic. Sudural haemorrhage can occur at any age but common at extremes of age. The condition is always due to trauma and there is probably no such entity as “spontaneous subdural hematoma”. As with Extradural, the position of Subdural can never be interpreted as a contre coup lesion and is thus of no use in differentiating a blow from a fall. It arises from torn communicating veins that traverse the subdural space between cortical vessels and the dural sinuses. Less often the sinuses themselves contribute to the haemorrhage. Chronic Subdural hematoma is found in old age as an incidental finding at autopsy, where death may be due to an unrelated cause. The recent lesion may be up to several weeks old which is tan or red-brown in color with gelatinous membrane covering the surface. The contents may be thick with few recent fresh bleeds. The old hematoma of months or years old is firmer and with thick surface. They contain liquid which is brown or straw colored. The chronic hematoma may become large and press down the cerebral hemisphere sufficiently to dent and distort the surface. The hematoma may eventually absorb or it may remain dormant at the same size or it may enlarge.

The mechanism of enlargement of subdural hematoma is controversial. One common explanation, which seems the most reasonable, is that it occurs from repeated future bleeding, perhaps from the new blood vessels that penetrate the mass as the part of healing process. The other theory involves osmosis, said to operate because the center of haemorrhage commonly

liquefies, forming the hemorrhagic fluid that osmotically attracts into it, the cerebrospinal fluid from outside the capsule, which acts as semipermeable membrane. The first mechanism seems more likely, as areas of fresh bleeding are often found inside a substantial hematoma but, whatever the cause, the final effect is worsening of space- occupying effect.

Subdural haemorrhage

Subarachnoid Hemorrhage

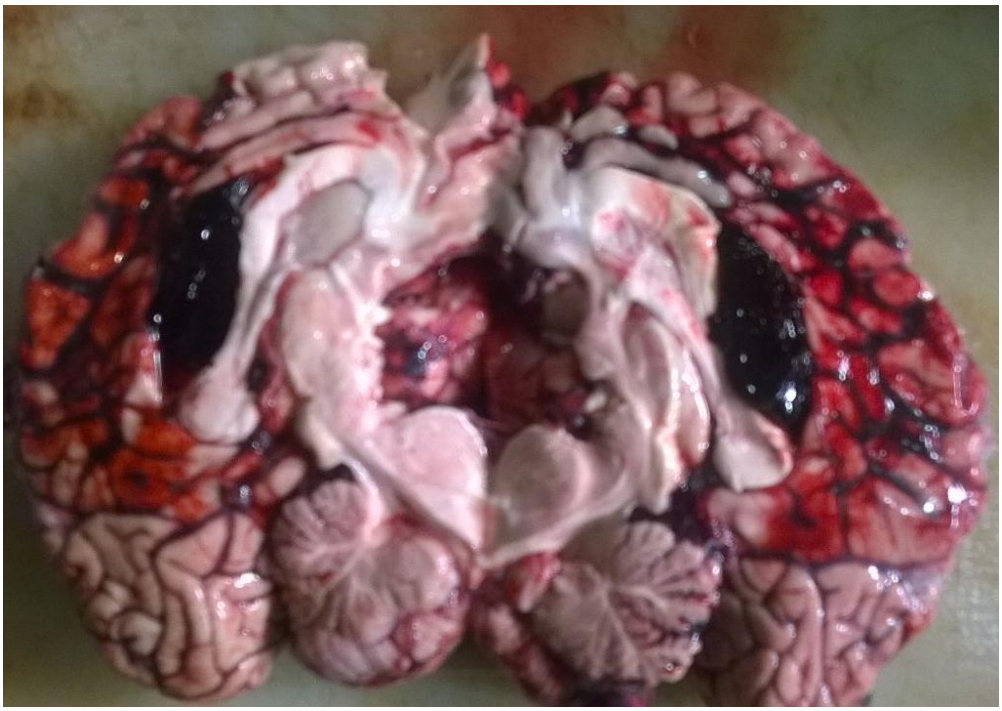


Subarachnoid haemorrhage.

Subarachnoid bleeding is more common than any other types of intracranial bleeding, but has a mixed etiology. It is uncommon for the traumatic subarachnoid bleeding to occur as pure lesion where there is no cortical contusion, no neck injury or deep brain injury. Slight Subarachnoid haemorrhages can occur after moderate trauma. It frequently can occur even in natural diseases. When trauma is also present, the complex association of either trauma precipitating the rupture or rupture causing fall or other accident leading to trauma has to be considered. When it is secondary for some laceration or cortical contusion, then it is localized and the severity depends on primary impact. Where it arises as a result of blunt impact, with or without other membrane bleeding or cortical bruising, its position is not a good localizing sign.

Blood of subarachnoid space mixes with cerebrospinal fluid, which dilutes it, makes it less ready to clot and allow more mobility. This bleeding from high over the cerebral hemispheres readily slides down to cover the brain and entire base of the brain. Bleeding in subarachnoid space is caused by shear stress and rotational movements of the brain. Rupture of the bridging veins that leaves cortex and penetrate the arachnoid en route for large draining veins and sinuses that lies in dura. Where there is laceration or contusions or infarction of the cortex bleeding will come from cortical veins and small arteries directly into the subarachnoid space. Traumatic subarachnoid bleeding can also arise from fracture of transverse process of first cervical vertebra, thus it damages

the artery contained in the tunnel like foramen in this bone leading to CV- One syndrome. It is uncommon instance when substantial subarachnoid haemorrhage seems to be the only sequel of a fatal head injury.



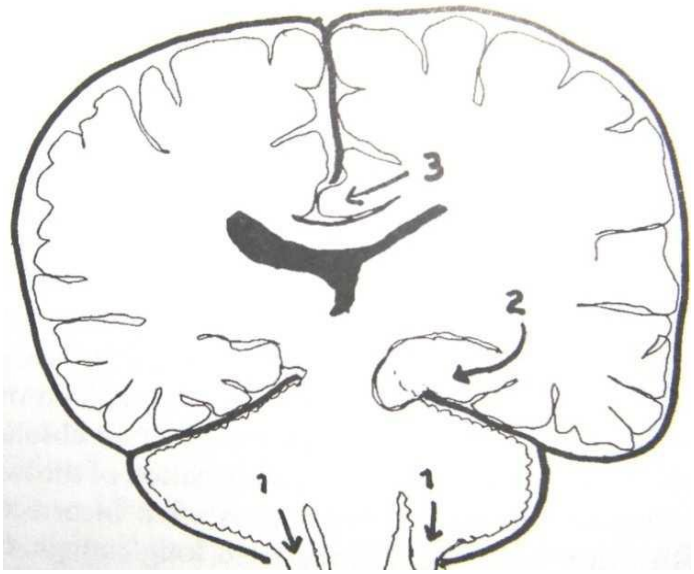
Intra cerebral haemorrhage

Intra cerebral haemorrhage

Cerebral oedema:

Swelling of the brain tissue is the local phenomena around almost any lesion like contusion, laceration, tumor, and infarct. Swelling of the brain is extremely common after sustaining head injury especially in children. Though it is an inevitable accompaniment of almost all intra-cerebral damages, either asgeneral or a local phenomenon, it may also occur as sole abnormality not infrequently proving fatal.

Cerebral Oedema



Oedema may be associated with diffuse neuronal injury or concussion. It is the most common cause for raised intracranial pressure. The amount of fluid

may increase and total weight may increase by atleast by 100gram, mainly in white matter. Autopsy findings like dura is stretched and tensed, the brain bulges through the first incision in the membranes. The gyri are pale and flattened, sulci filled (narrowed) giving the normal corrugated cerebral surface the appearance of smoothness. The ventricles may also be reduced in size. Severe cerebral oedema causes largervolume cerebral hemispheres to press down upon the tentorium and herinate through midbrain opening. In the absence of any other demonstrable lesions, cause of death can be attributed to the swelling of the brain, compressing the vital centers of the brainstem.

Concussion:

Concussion is a clinical entity, and not a pathological entity. But it must be considered as it is related to the intracranial lesion. Concussion is defined as the disorder of cerebral function which follows upon impact of a force to the head. It is extremely common, but not inevitable sequel to any significant mechanical insult to the brain. Though the duration is related to the severity, the concussion may be a small event that the patient may not even fall to the ground. True concussion may last for seconds or minutes. If prolonged unconsciousness extends into hours or, days or longer, then there is likely to be some structural damage.

Road Traffic Accidents:

Road traffic accidents are important causes of mortality and morbidity in the world. With the expansion in road network, motorization and urbanization

in the country, the number of road accidents have surged. RTAs and fatalities have emerged as a major public health concern, with RTAs having become one of the leading causes of deaths, disabilities and hospitalizations which impose severe socio-economic costs across the world The United Nations has rightly proclaimed 2011-20 as the decade of action on road safety and have called upon all member countries to prepare a decadal action plan for implementation in their respective countries so that the present rising trend of road accidents stabilizes and is reversed by the year 2020.

WORLD:

According to WHO estimates[1], about 1.24 million people die each year as a result of road traffic crashes. Between 20 to 50 million more people suffer non-fatal injuries, with many incurring a disability as a result of their injury. Road traffic injuries are the leading cause of death among young people, aged 15–29 years.

91% of the world's fatalities on the roads occur in low-income and middle-income countries, even though these countries have approximately half of the world's vehicles. Half of those dying on the world’s roads are “vulnerable road users”: pedestrians, cyclists and motorcyclists. Without action, road traffic crashes are predicted to result in the deaths of around 1.9 million people annually by 2020.

Only 28 countries, representing 416 million people (7% of the world's population), have adequate laws that address all five risk factors (speed, drink-driving, helmets, seat-belts and child restraints).

COST:

Road traffic injuries cause considerable economic losses to victims, their families, and to nations as a whole. These losses arise from the cost of treatment .(including rehabilitation and incident investigation) as well as reduced/lost productivity (e.g. in wages) for those killed or disabled by their injuries, and for family members who need to take time off work (or school) to care for the injured.

There are few global estimates of the costs of injury, but an estimate carried out in 2000 suggest that the economic cost of road traffic crashes was approximately US$ 518 billion. National estimates have illustrated that road traffic crashes cost countries between 1–3% of their gross national product, while the financial impact on individual families has been shown to result in increased financial borrowing and debt, and even a decline in food consumption.

INDIA:

According to Ministry Of Road Transport and Highways (MORTH)[2], during the year 2010, there were around 5 lakhs road accidents, which resulted

in deaths of 134,513 people and injured more than 5 lakhs persons in India. These numbers translate into 1 road accident every minute, and 1 road accident death every four minutes. 1.5 The loss to the Indian economy due to fatalities and accident injuries estimated at 3% of GDP in 1999-2000 is particularly severe as 53.1% of road accident victims were in the age group of 25 to 65 years in 2010, with pedestrians, bicyclists and two-wheelers, who comprise the most unprotected road users, accounting for around 40% of all fatalities. Number of accidents per lakhs population increased from 21.2 in 1970 to 42.5 in 2010.Road accidents in the country have decreased by around 40% during 2016,with the year seeing 4,80,652 road accidents as against 5,01,423 in 2015.However fatalities resulting from these accidents have risen by 3.2% during the same period .Nearly 1,50,785 persons were killed in 2016 as against 1,46,133 in 2015.

TAMILNADU:

The top five States in India in total number of road accidents, persons killed and persons injured in road accidents Maharashtra, Tamilnadu, Madhya Pradesh, Karnataka and Andhra Pradesh.

Despite claims of Tamil Nadu adopting modern road safety measures, the number of deaths on roads has gone up in the state. According to statistics compiled by the transport department and the state police, 15,900 people were killed in road accidents in 2012, up from 15,422 the previous year. This means at least 43 people die in accidents every day[13].

RISK FACTORS

Young adults aged between 15 and 44 years account for 59% of global road traffic deaths. More than three-quarters (77%) of all road traffic deaths occur among men.

An increase in average speed is directly related both to the likelihood of a crash occurring and to the severity of the consequences of the crash. Pedestrians have a greater chance of surviving a car crash at 30 km/hour or below.30 km/hour speed zones can reduce the risk of a crash and are recommended in areas where vulnerable road users are common (e.g. residential areas, around schools).

Drinking and driving increases both the risk of a crash and the likelihood that death or serious injury will result.The risk of being involved in a crash increases significantly above a blood alcohol concentration (BAC) of 0.04 g/dl.

Wearing a motorcycle helmet correctly can reduce the risk of death by almost 40% and the risk of severe injury by over 70%.

Wearing a seat-belt reduces the risk of a fatality among front-seat passengers by 40–50% and of rear-seat passengers by between 25–75%.

If correctly installed and used, child restraints reduce deaths among infants by approximately 70% and deaths among small children by between 54% and 80%.

There are many types of distractions that can lead to impaired driving, but recently there has been a marked increase around the world in the use of mobile phones by drivers that is becoming a growing concern for road safety.

Drivers using a mobile phone are approximately four times more likely to be involved in a crash than when a driver does not use a phone. Hands-free phones are not much safer than hand-held phone sets.

A systematic review of available literature on the use of psychoactive substances (alcohol and drugs) among road users, particularly those involved in road traffic accidents in India was done by Das A et al in 2012[14]. 23 studies were included in the review, alcohol was reported by all, but only 2 mentioned the use of drugs. Most of the studies were hospital based, included injured or killed road users, and belonged to southern parts of India. It was found that 2 to 33 percent of injured and 6 to 48 percent of killed RTA victims had consumed alcohol or drugs.

With the aim of exploring various epidemiological characteristics of RTAs, a retrospective analysis of medico-legal autopsies was conducted by Kanchan et al[15] between January 2005 and December 2009 in the Department of Forensic Medicine, Kasturba Medical College, Manipal in Karnataka, South

India. Out of the 879 autopsies conducted during the study period, 39% were due to RTAs. Among the victims, 89.8% were males and 10.2% were females. The mean age of victims was 38.7 years, which was slightly higher in females compared to males. Most of the male victims belonged to the age group 20-29 years. The head injuries were responsible for nearly 3/4th of deaths followed by abdominal injuries (6.7%). The mean duration of survival following road traffic accident was 6-7 days. Occupants of motorized two wheelers (43%) and pedestrians (33%) were the most common victims of RTAs followed by occupants of light motor vehicles (LMVs). The most common offending agents in road traffic accidents were heavy motor vehicles (35.2%) followed by light motor vehicles (31.7%). It was concluded that RTAs are important public health hazards and should be addressed through strengthening of emergency healthcare, stricter enforcement of traffic laws and health education.

In a study done by Bayan et al[16] in Pune, a total of 212 non-fatal road traffic accidents admitted over a period of one year were analysed. Male: female ratio was almost 5:1 ($P < 0.0001$). The maximum accidents occurred on Sundays and Mondays and the least around midweek (Wednesday) ($P < 0.001$). Pedestrians were the most vulnerable group, followed by drivers and pillions of two wheelers. Accidents were more likely in the time zone of 8 pm to midnight, followed by 4 pm to 8 pm ($P < 0.0001$). A majority of the patients sustained multiple injuries followed by injuries to the lower limbs. A majority reported impaired visibility and fatigue as the cause of accident.

In a cross-sectional study conducted Sreedharan et al[17] in Kerala, India, over a period of six months, 309 motorcyclists in Kerala were interviewed for this study using a pretested structured questionnaire. It was found that among the total, only 31.4% used a helmet.

PATTERN OF INJURIES IN RTAs:

In a study done by Menon et al[18] 682 victims of road traffic accidents who died due to injuries sustained to the head were autopsied at District Wenlock Hospital, Mangalore, India over a period of 5 years between January 1999 and December 2003. Skull fractures were present in 88.88% of the cases. Fractures of the vault were found in 88%, base of the skull in 35.97% and a combination of both in 35% of cases. In most of the cases, fissured fractures were found (23%). Among intra-cranial haemorrhages, subdural haemorrhage was found in 52.63% and subarachnoid haemorrhage in 27.27% of cases. Contusions and lacerations of brain were found equally in 35% of cases.

In a study done by Rasouli et al[19] in Iran, 64 patients with spinal cord injury due to road traffic accidents were studied. 31 patients (81.6%) had complete spinal cord injury. Conusmedularis (T12-L2) was the most affected level.

In a study done by Kortar et al[20] in United Kingdom on a total of 429 motorcycle accident victims, lower limb injuries represented the commonest type of injury (238, 55.5%). Fractures were the commonest lower limb injuries (73.4%) and closed fractures were commoner than the open fractures. The commonest anatomical location of fractures in lower limbs was tibial shaft. It was concluded that lower limb injuries represent the commonest form of injuries among the motorcycle accident victims. Fractures were the commonest type of injury seen and the most common location was shaft of tibia.

In a study done by Akama et al[21] in Kenya on the pattern of maxillofacial and associated injuries in road traffic accidents, Four hundred and thirteen (85.7%) had non-fatal injuries whereas 69 (14.3%) had sustained fatal injuries. Males in the 21-30-year age group were the most affected. In the fatal category head injury alone was the leading cause of death accounting for 37.7% of the cases, followed by head and chest injuries combined which were responsible for 13% of the cases. Only 5.1% of the casualties with non-fatal injuries had fractures involving the maxillofacial skeleton. Skeletal injuries other than those involving the maxillofacial region were found in 142 (34.1%) incidents. In the non-fatal category 89.6% of the casualties had soft tissue injuries (STIs) involving the craniofacial region with facial cuts being the majority (69.2%). Two hundred and seventy three (66.1%) incidents of other STIs than those of the head region were noted, the lower limbs accounting for 45.4% of these.

In a study done by Ogleznev et al[22] on cranio cerebral trauma in road traffic accidents, the most severe form of brain compression was multifactorial compression (27.6%) and its most common form was compression with subdural hematoma (35.3%). In over half the cases (62.6%), BI due to RTA was associated with extra cranial lesions, leading to diagnostic problems. The pattern and site of lesions were related to the type of a transport vehicle and to the role of a victim as a traffic participant. Multiple extra cranial lesions were mostly frequently encountered in victim pedestrians (30.3%), BI concurrent with chest damage was common in drivers (12.8%), BI concurrent with "whip" injury of the cervical spine was found in drivers and passengers though such combinations were also seen in pedestrians (1.5%-5 cases).

In a study done by Jacobsen et al[23] in Copenhagen, a total of 428 cases of cranio cerebral trauma due to various causes were analysed. Among the 428 cases with skull fractures, linear fracture was the commonest type (251), followed by comminuted (62), depressed (22), ring (15) and spider web (3) fractures. Multiple fracture types were found in 22 cases, combination of fracture types in 42 cases and the fracture type was inconclusive in 11 cases. Totally 998 intracranial leisons were found in all the cases. Subarachnoid haemorrhage was found in 395 cases, Extradural haemorrhage in 20 cases, Subdural hemorrage in 98 cases, contusions of cerebrum and cerebellum in 320 cases, lacerations of cerebrum and cerebellum in 113 cases, intraparenchymal hemorrhages in 52 cases.

In a study done by Oberai et al[24] in Patiala, India, pattern of injuries in road traffic accidents involving two-wheelers were analysed. The maximum deaths in fatal two wheeler accidents were due to head/face injuries 33(66%) followed by chest injuries 6(12%) and abdominal injuries 4(8%). Minimum number of deaths occurred due to combined injuries of head and chest 1 (2%) followed by combined injuries of head and abdomen 2 (4%), followed by pelvis 2 (4%) and lower limb 2 (4%) respectively.

OBSERVATION AND

RESULTS

OBSERVATION AND RESULTS

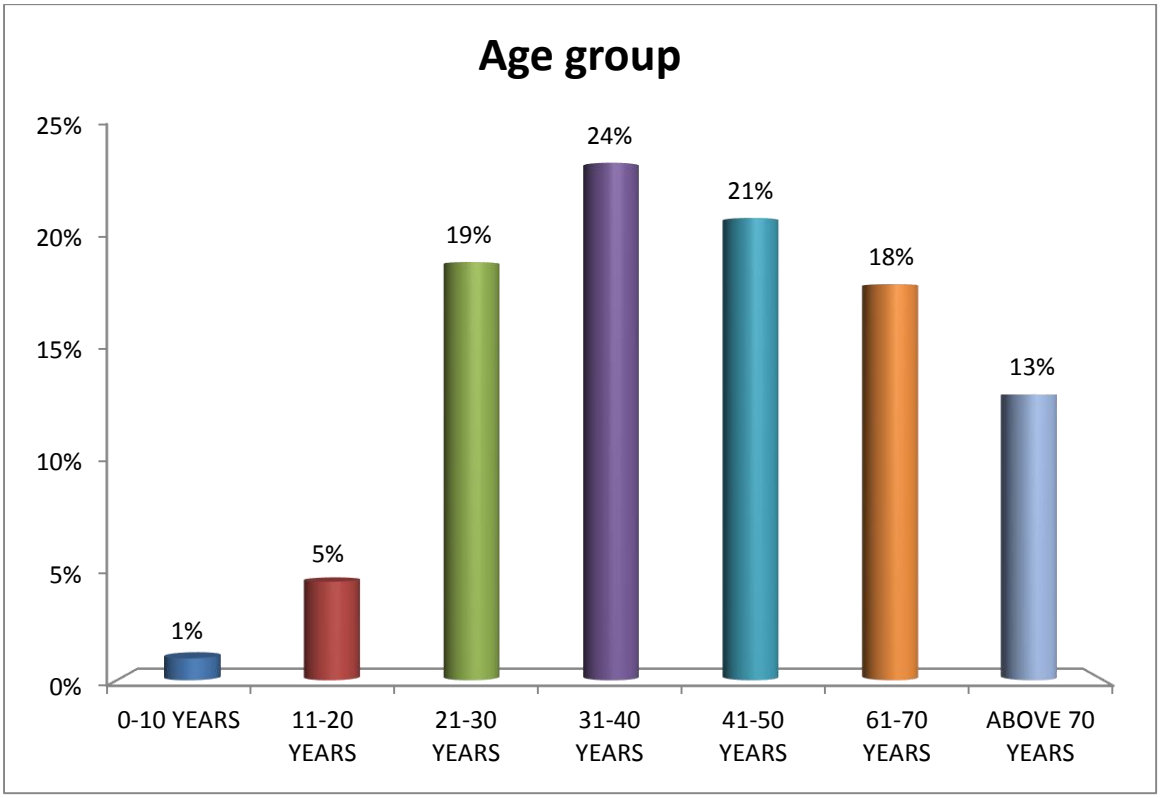
The present study of pattern of intracranial hemorrhages in fatal head injury cases of road traffic accident was carried out in the Institute of forensic medicine, Rajiv Gandhi Government General Hospital, Chennai-3. 200 cases were autopsied over a period of 1 year from August 2016 to August 2017.

The Data has been systematically recorded and various observation were made and tabulated in tables.

Table-1 Agewise distribution of cases.

AGE_GROUP	Frequency	Percent
0-10 YEARS	2	1.0
11-20 YEARS	9	4.5
21-30 YEARS	38	19.0
31-40 YEARS	47	23.5
41-50 YEARS	42	21.0
61-70 YEARS	36	18.0
ABOVE 70 YEARS	26	13.0
TOTAL	200	100.0

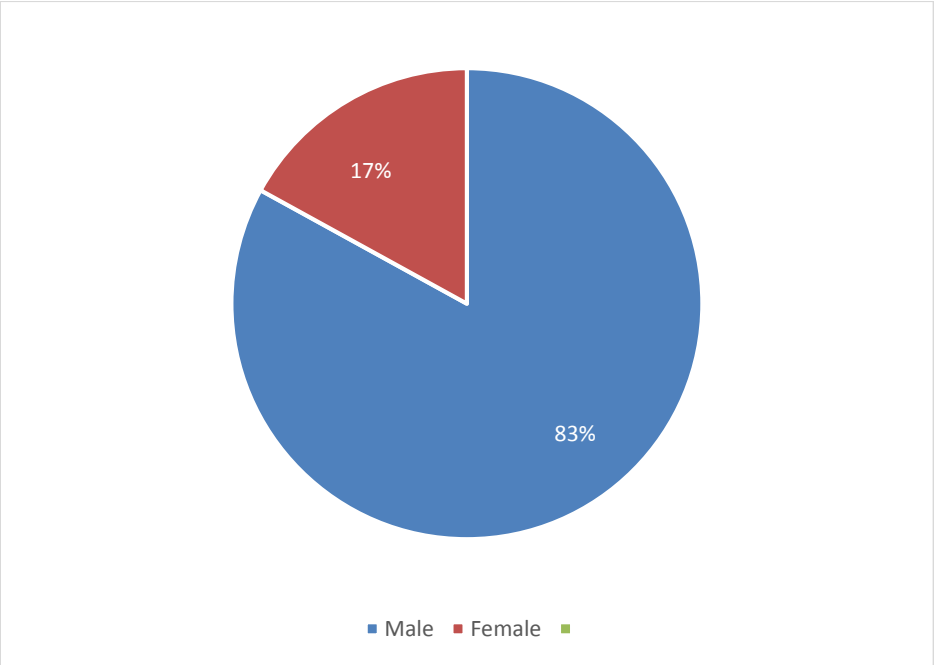
As in the above table 1 ,the most vulnerable age group was those in 31- 50 years followed by the age group of 21 – 30 years .The obvious reason being that they form the most active group of the society and hence are prone to road traffic accidents .



Age Wise Distribution of RTA cases

Table -2 Sex wise distribution of fatal RTA CASES.

SEX	Frequency	Percent
MALE	166	83.0
FEMALE	34	17.0
Total	200	100.0



As in the Table -2 (83%) Were males and (17%) were females, males were more prone to head injuries in road traffic accidents since they are more into outdoor activities like driving vehicles ,working outdoor posing them risk due to accidents.

Females succumbed to road traffic accidents were mainly due to they being pillion riders without helmet and pedestrians.

Table -3 Pattern of Scalp contusion among fatal RTA CASES.

Scalp Contusion	Frequency	Percent
BOTH FTP	7	3.5
BOTH PARIETO OCCIPITAL	1	.5
DIFFUSE	79	39.5
LT CEREBRAL	1	.5
LT FTP	40	20.0
LT FTP AND OCCIPITAL	3	1.5
MID PARIETAL	1	.5
NO	10	5.0
OCCIPITAL	1	.5
RT FTP	55	27.5
RT PARIETAL	2	1.0
Total	200	100.0

As in the above table 3 out of the 200 cases studied scalp contusion was present in 190 cases. Diffuse scalp deep contusion of the scalp was commonest in 79 cases (39.5%) followed by scalp contusion of right fronto tempero parietal region of scalp.

Table -4 Incidence of scalp laceration

LACERATION	Frequency	Percent
YES	20	10.0
NO	180	90.0
Total	200	100.0

As seen in the above table 4 laceration of the scalp was present in 20 cases i.e., 10% of cases

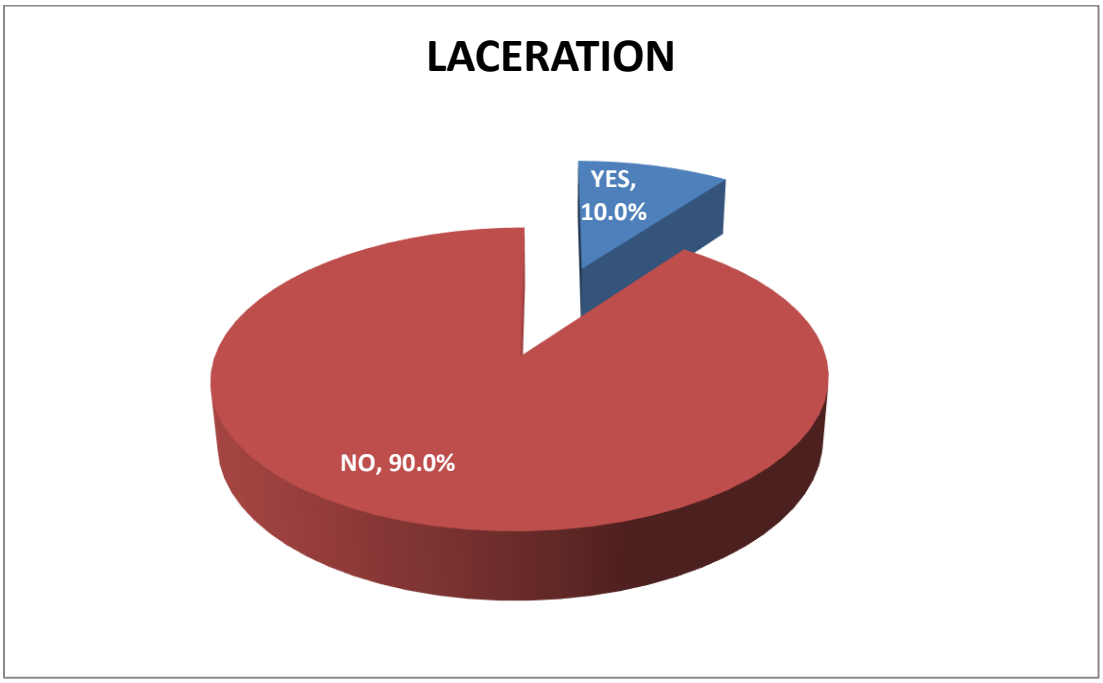


Table-5 Percentage of different types of fracture of the skull.

FRACTURE	FREQUENCY	PERCENTAGE
BONY AND DURAL DEFECT	23	11.5
COMMINUTED	8	4
DIASTATIC	1	0.5
DEPRESSED	6	3
INTACT	81	40.5
FISSURE	81	40.5

As in the above table linear fractures were the commonest type (81) Cases 40.5% followed by Comminuted fracture 8 cases 4.0% and depressed fracture in 6 cases .i.e 3.0%.

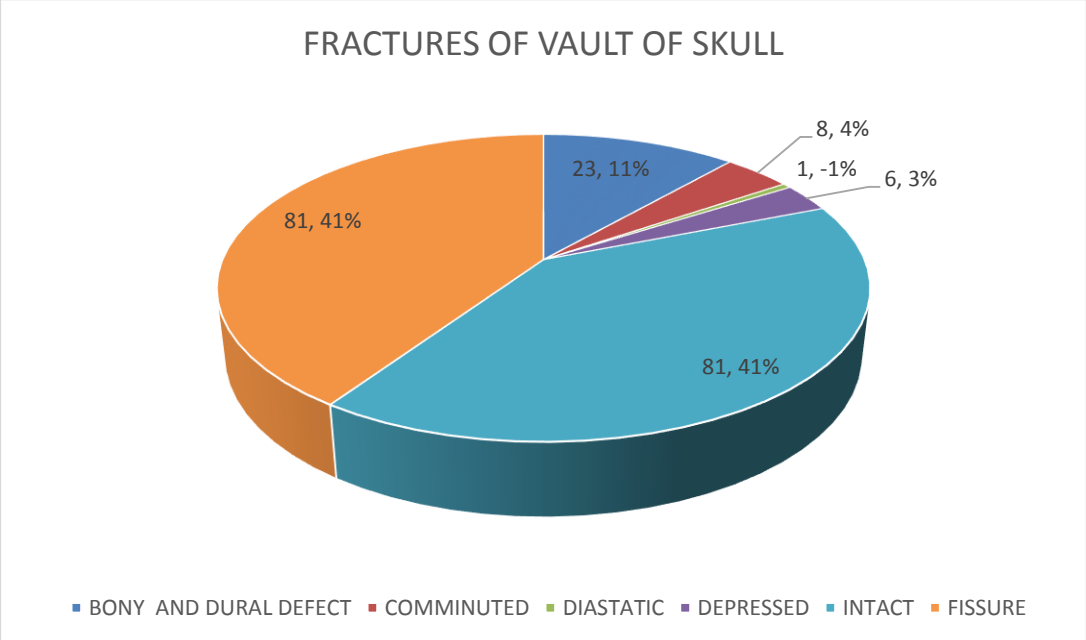


Table -6 Region wise distribution of extradural hemorrhage among fatal RTA CASES.

EDH	FREQUENCY	PERCENTAGE
PARIETAL	19	9.5
FRONTAL	2	1
TEMPORAL	6	3
OCCIPITAL	1	0.5
NIL	172	86

As in the above table 6- extradural haemorrhage was more commonest in parietal region 19 cases 9.5% followed by temporal region 3% and least common in frontal 1% and occipital regions.0.5%

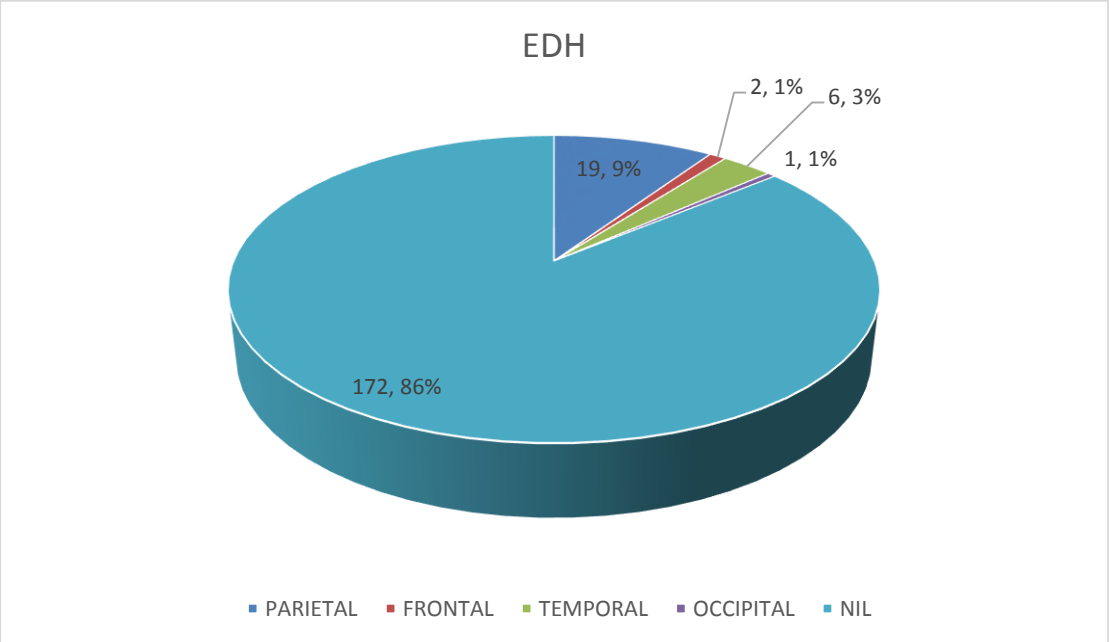


Table 7.Distribution of Subdural hemorrhage in fatal head injury cases in RTA

SDH	Frequency	Percent
RTFTP	37	18.5
LTFTP	34	17.0
DIFFUSE	119	59.5
NO	10	5.0
Total	200	100.0

As in the above table the most common extra axial haemorrhage was subdural haemorrhage. Subdural haemorrage was more common in both the cerebral and cerebellar hemispheres of brain .59.5% followed by the surface of fronto temperoparietal region of right cerebral hemisphere of brain 37 cases (18.5%).

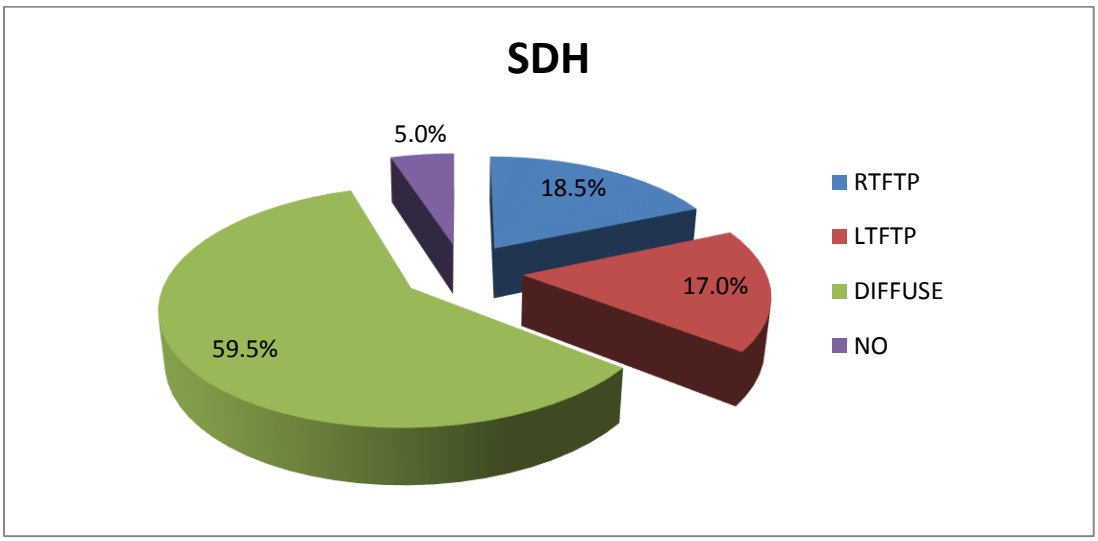


Table-8 Distribution of subarachnoid hemorrhage in fatal head injury cases.

SAH	Frequency	Percent
RT FTP	33	16.5
LT FTP	25	12.5
DIFFUSE	111	55.5
NO	31	15.5
Total	200	100.0

From the above table 8 - It is obvious that subarachnoid haemorrhages were present in 169 cases. The most common site for subarachnoid haemorrhage was on the surfaces of both the cerebral and cerebellar hemispheres of brain 55.5% followed by the surface of fronto tempero parietal region of the right cerebral hemisphere of brain. 16.5%.

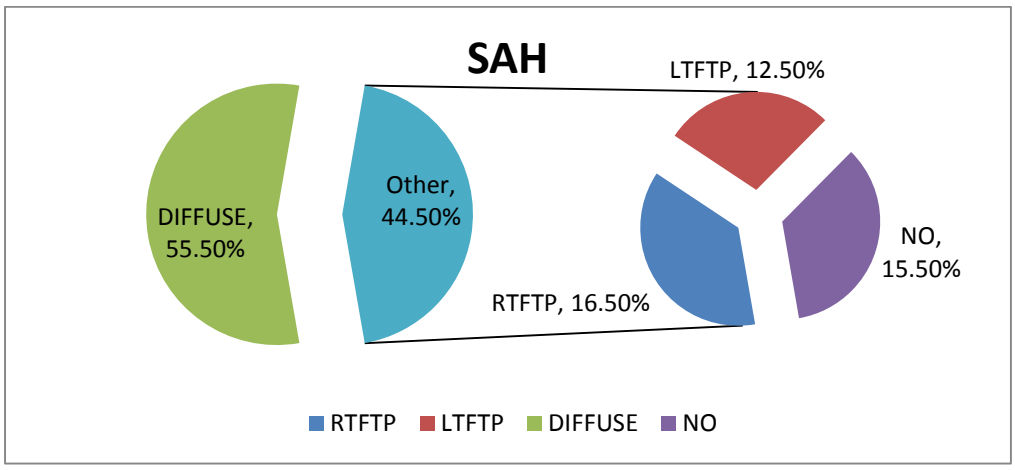


Table -9 –Incidence of intracerebral hemorrhage in fatal head injury cases in RTA

ICH	Frequency	Percent
PRESENT	19	9.5
ABSENT	181	90.5
Total	200	100.0

From the above table -9 it is evident that out of the 200 cases studied only in 19 cases i.e 9.5% of cases intracerebral haemorrhage was present.

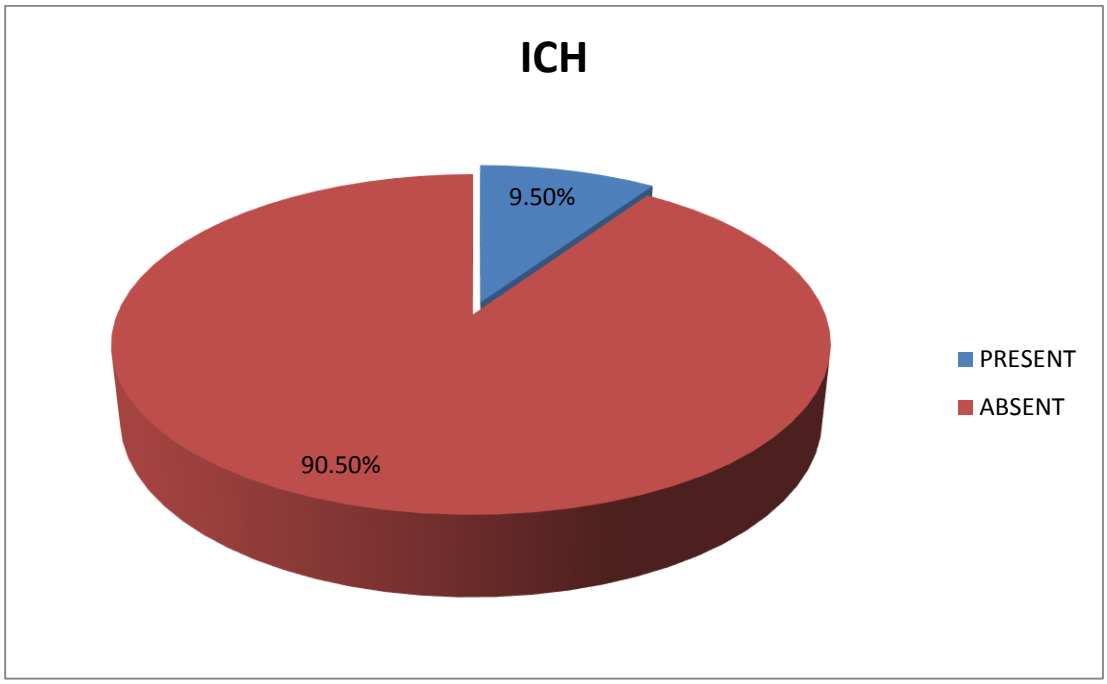


Table -10-Incidence of intraventricular hemorrhage in fatal head injury cases of RTA

IVH	Frequency	Percent
PRESENT	27	13.5
ABSENT	173	86.5
Total	200	100.0

From the above table 10 - it is obvious that intraventricular haemorrhage was present in only 27 cases i.e 13.5%

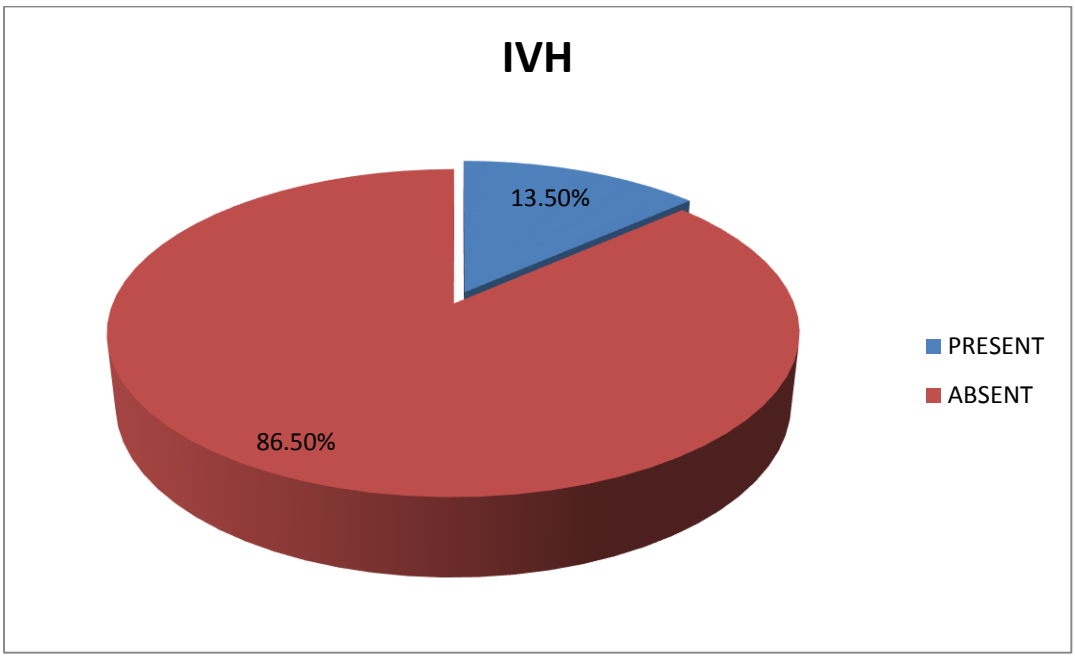
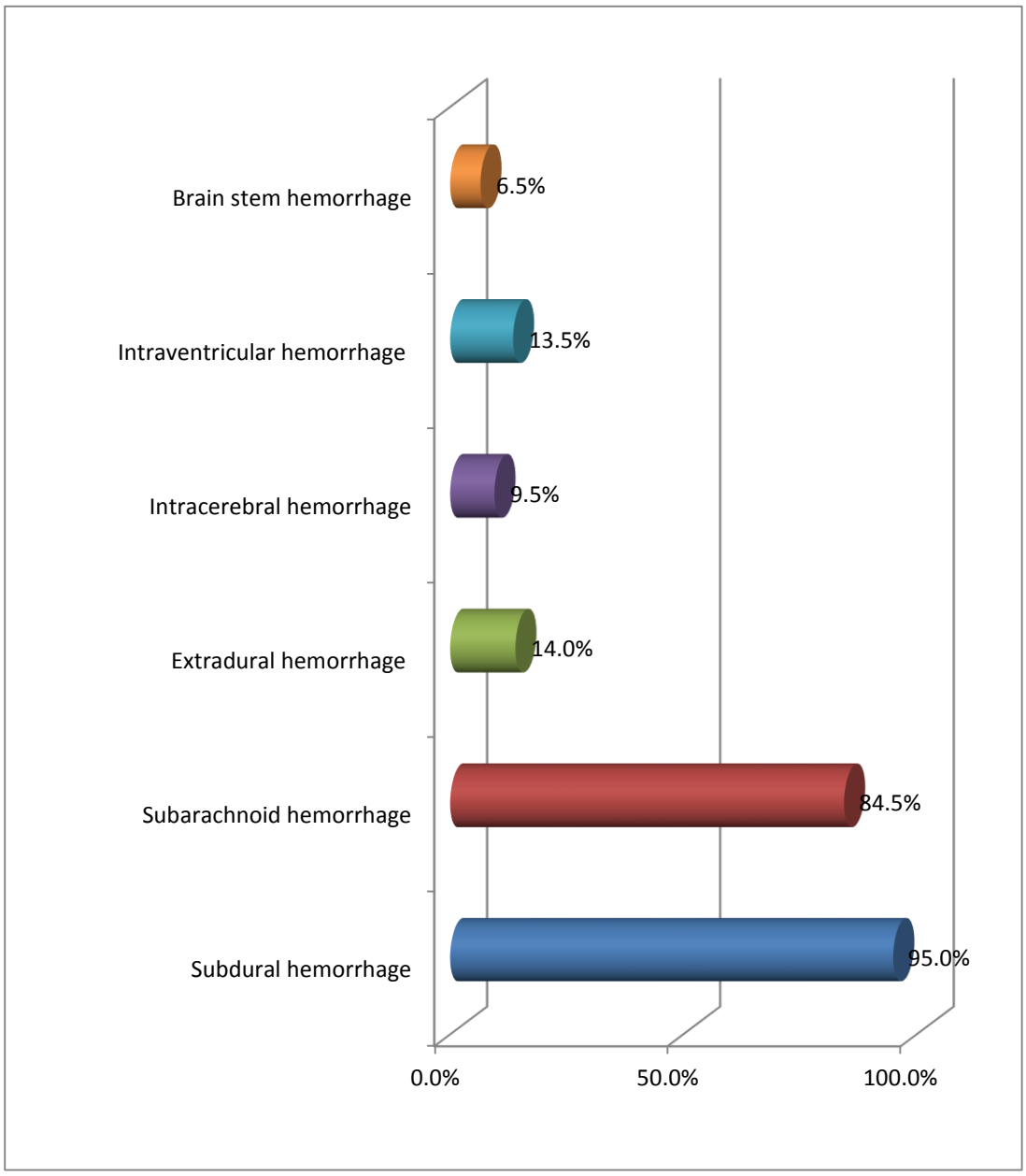


Table -11- Percentage and Type of Intracranial hemorrhages in fatal head injury cases of RTA

	FREQUENCY	PERCENT
Subdural hemorrhage	190	95.0%
Subarachnoid hemorrhage	169	84.5%
Extradural hemorrhage	27	13.5%
Intra cerebral hemorrhage	19	9.5%
Intra ventricular hemorrhage	27	13.5%
Brain stem hemorrhage	13	6.5%

As in the above table -11 –Subdural hemorrhage was present in 190 cases 95%, followed by subarachnoid hemorrhage 84.5%, extradural hemorrhage 13.5%, intra ventricular hemorrhage 13,5%, intra cerebral hemorrhage 9.5% and brain stem hemorrhage 6.5%

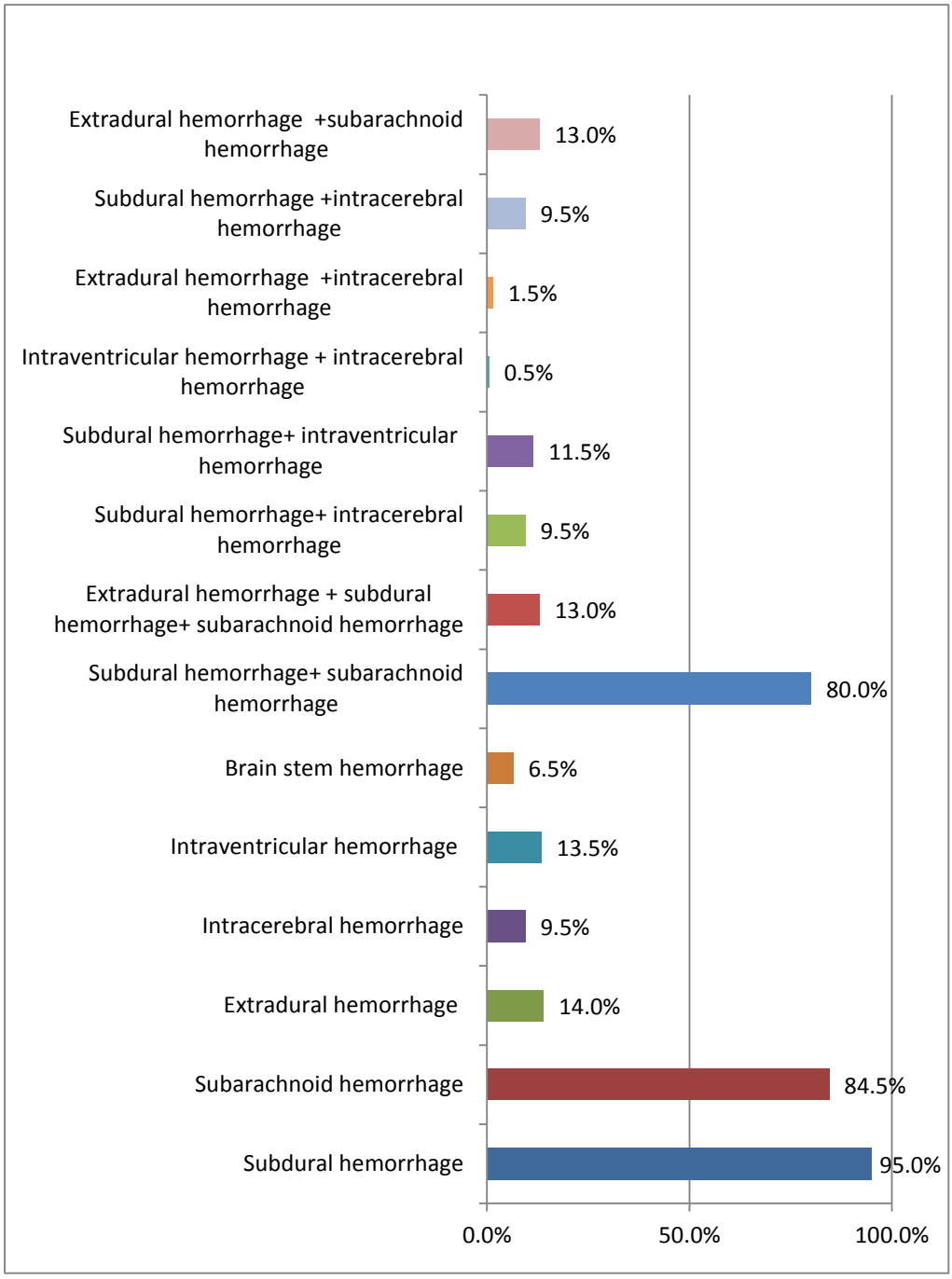


Percentage and Type of Intracranial hemorrhages in fatal head injury cases of RTA

Table -12-Types and Percentage of Combination of intracranial hemorrhage in head injury cases of RTA

	FREQUECNY	PERCENT
Subdural hemorrhage	190	95.0%
Subarachnoid hemorrhage	169	84.5%
Extradural hemorrhage	28	14.0%
Intra cerebral hemorrhage	19	9.5%
Intra ventricular hemorrhage	27	13.5%
Brain stem hemorrhage	13	6.5%
Subdural hemorrhage+ subarachnoid hemorrhage	160	80.0%
Extradural hemorrhage + subdural hemorrhage+ subarachnoid hemorrhage	26	13.0%
Subdural hemorrhage + intra cerebral hemorrhage	19	9.5%
Subdural hemorrhage + intra ventricular hemorrhage	23	11.5%
Intra ventricular hemorrhage + intra cerebral hemorrhage	1	0.5%
Extradural hemorrhage +intra cerebral hemorrhage	3	1.5%
Subdural hemorrhage+ intra cerebral hemorrhage	19	9.5%
Extradural hemorrhage + subarachnoid hemorrhage	26	13.0%

From the above table12, it is obvious that if type of intra cranial hemorrhage is considered in isolation, then cases having subdural haemorrhages were the highest in number 190 cases (95%) followed by cases of subarachnoid haemorrhages 169 cases (84.5%),extradural haemorrhages 14 and intra cerebral haemorrhages.9.5%.



Types and Percentage of Combination of intracranial hemorrhage in head injury cases of RTA

Table -13-Incidence of contributed factors

CONTRIBUTORY FACTORS	Frequency	Percent
ALCOHOL	5	2.5
NO	195	97.5
Total	200	100.0

As in the above table -13 alcohol contributed to 2.5%.

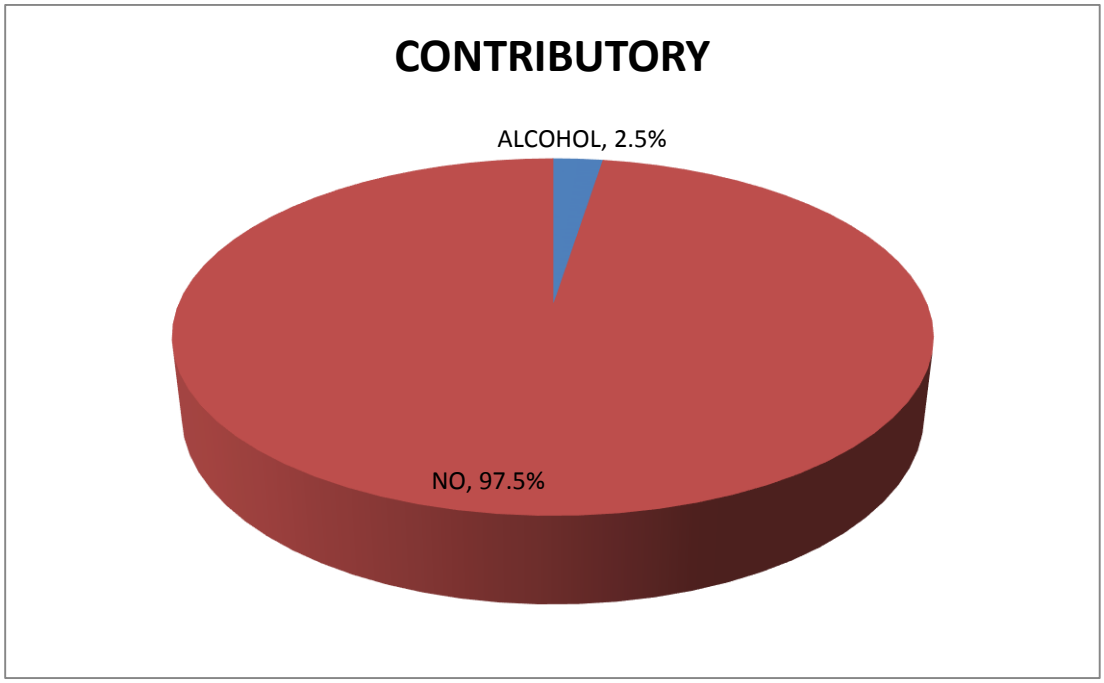


Table -14- Incidenc of brain stem hemorrhage in fatal head injury cases in RTA.

BRAINSTEM HEMORRHAGE	Frequency	Percent
YES	17	8.5
NO	183	91.5
Total	200	100.0

From the above table 14 it is evident that brain stem haemorrhage was present in 17 cases.(8.5%)

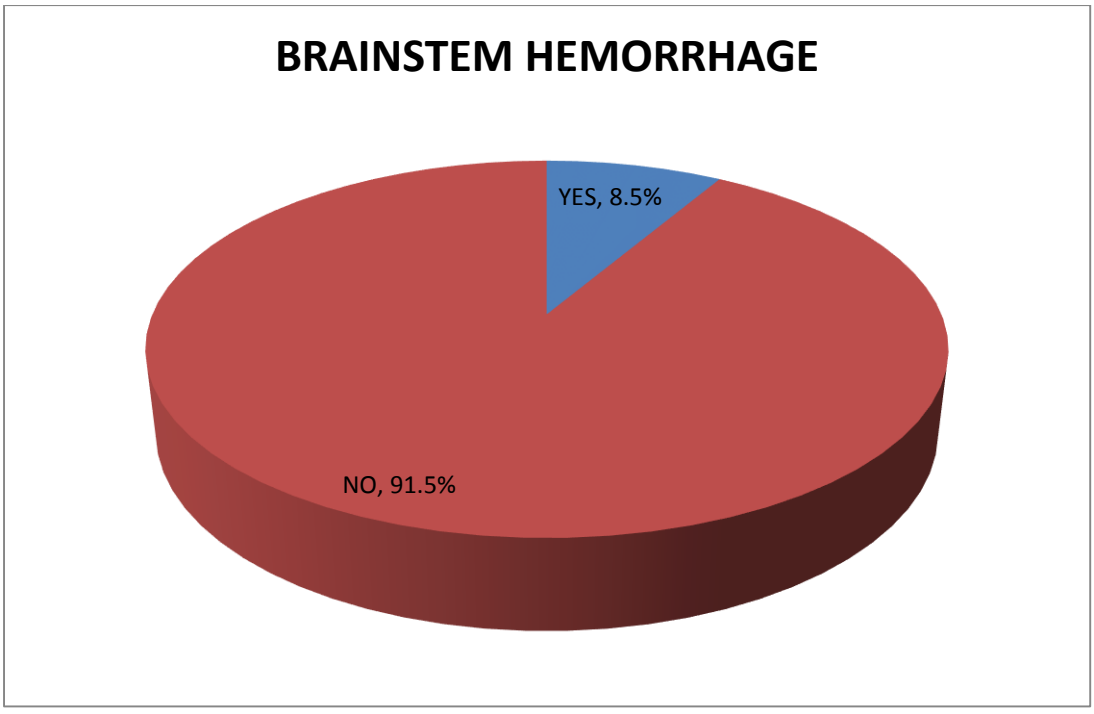


Table -15-Percentage and Type of Victims in Head Injury cases in RTA

DRIVER / OCCUPANT / PEDESTRAN	FREQUECNY	PERCENT
2 WHEELAR DRIVER	80	40.0%
2 WHEELER PILION RIDER	19	9.5%
PEDESTRAN	80	40.0%
OTHERS	21	10.5%
TOTAL	200	100.0%

As shown in the above table 15, among the type of road users, two wheeler drivers accounted for 80 cases 40%, pedestrians accounted for 80 cases 40% followed by two wheeler pillion riders 19 Cases (9.5%)

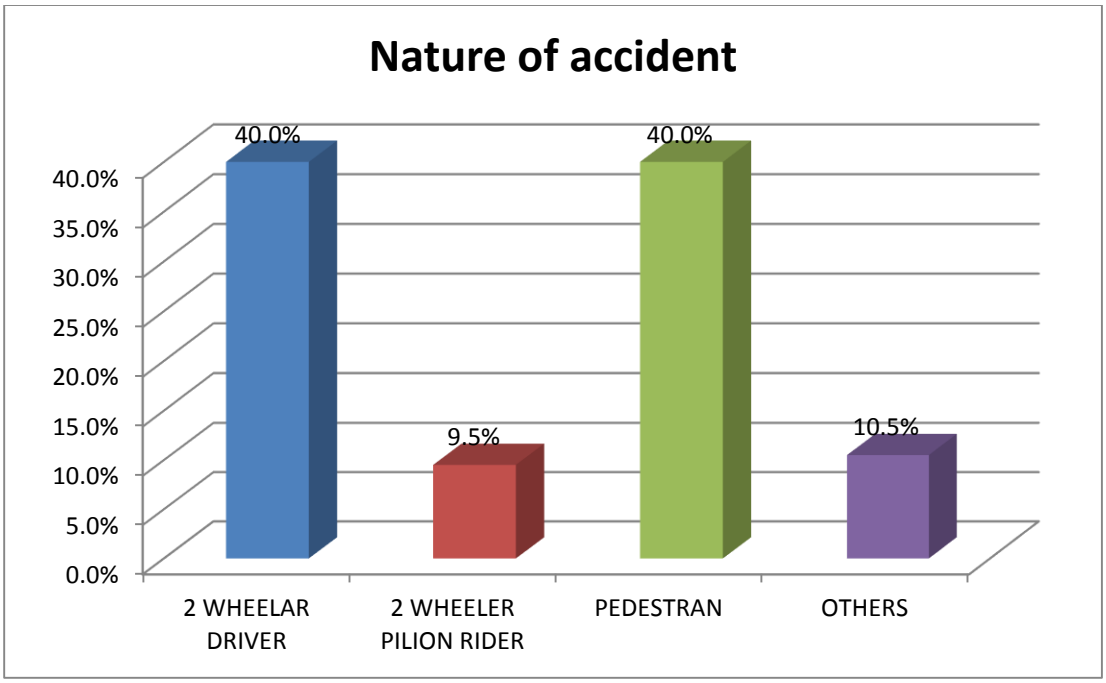


Table -16- Percentage of Mode of Accident

MODE_OF_ACCIDENT	FREQUECNY	PERCENT
2 WHEELER VS PEDESTRAN	53	26.5%
2 WHEELER VS 2 WHEELER	17	8.5%
SELF FALL	49	24.5%
OTHERS	81	40.5%
TOTAL	200	100.0%

As shown in the above table 16 as far as the mode of accident was considered two wheeler vs pedestrian accounted for 26.5% of cases followed by skid and fall from two wheeler 17 cases 8.5%

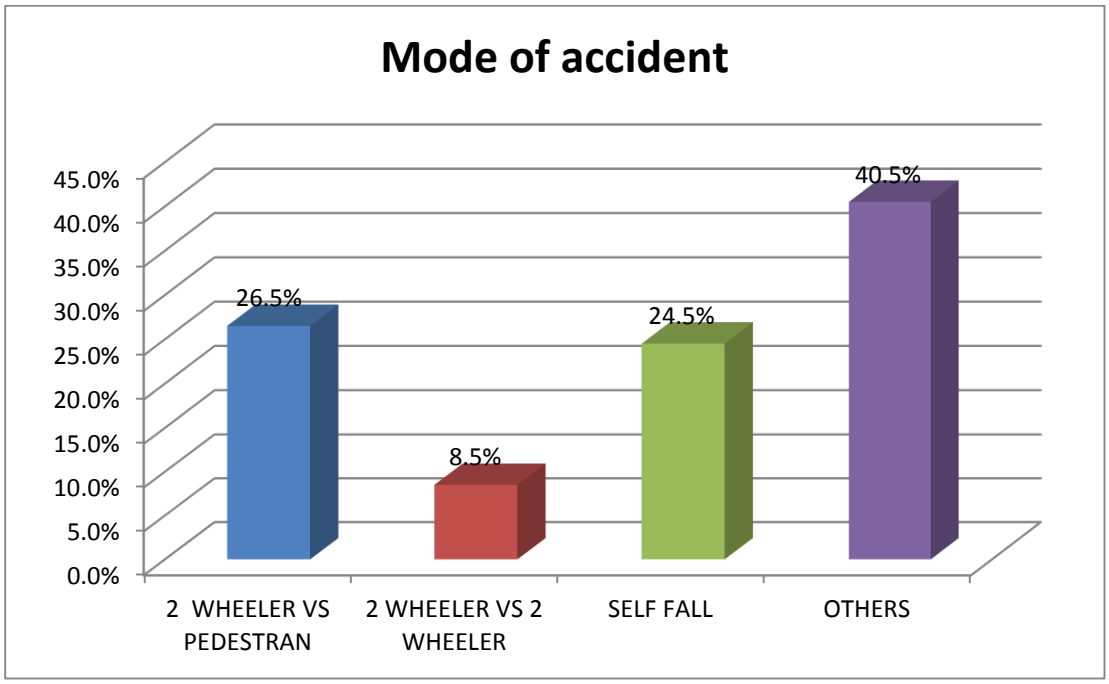
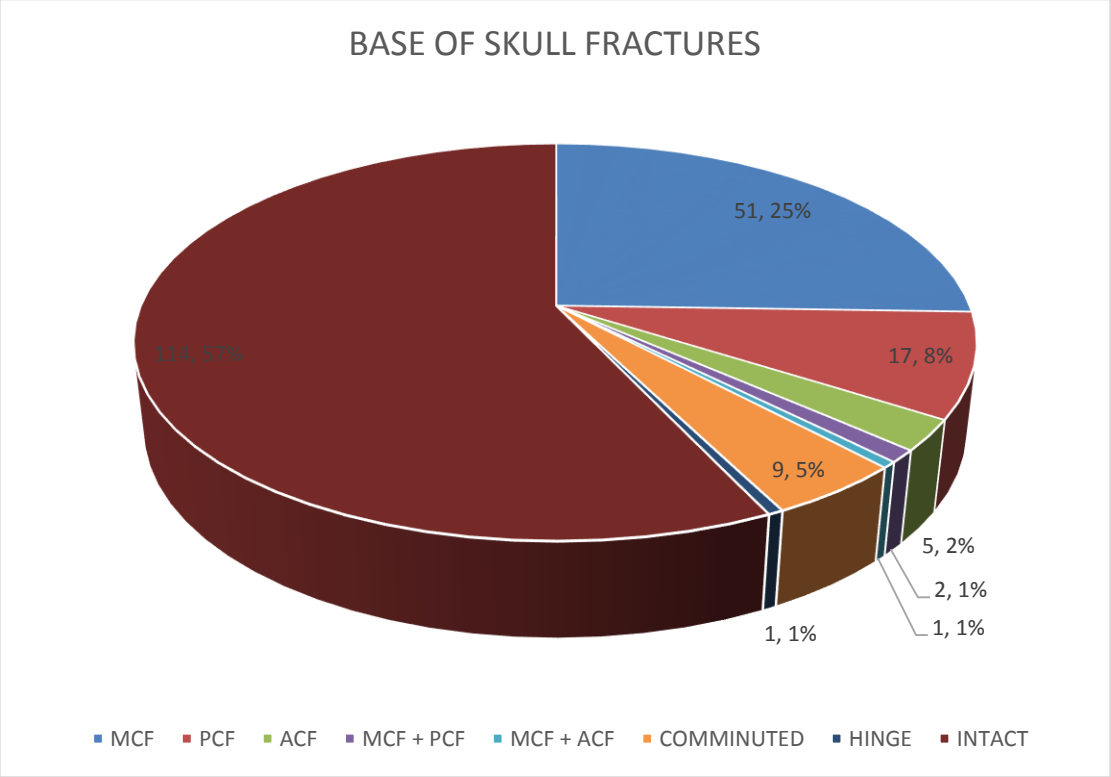


Table -17-Percentage and site of fracture of base of skull

MCF	51	25.5
PCF	17	8.5
ACF	5	2.5
MCF + PCF	2	1
MCF + ACF	1	0.5
COMMINUTED	9	4.5
HINGE	1	0.5
INTACT	114	57
TOTAL	200	100

From the above table 17 it is observed that fracture of the base of the skull 86 cases. middle cranial fossa is the commonest 51 cases. (23.5%) Fracture of the posterior cranial fossa is observed in 17 cases only (8.5%).



DISCUSSION

DISCUSSION

This study was done for a One years at Institute of forensic medicine. Madras Medical College .Chennai -03. Head injuries are one of the leading causes of mortality and morbidity in the world.

200 cases were studied.

Head injury can occur at any age.

In our study, as in **Table** 1 the most vulnerable victims were in the 31-50 years age group 44.5%, followed by the age group of 21 -30 19 % years, as in any other study . The obvious reason being they form the mainstream working group and hence are more prone to RTAs. 83 % of the victims in our study were males, thus constituting more than three-fourths of the total number of cases. This is because men are more into outdoor activities like driving vehicles posting them risk due to road traffic accidents, whereas females succumbed to RTAs mainly when they are pillion rider or pedestrians.

According to WHO[1] estimates, young adults aged between 15 and 44 years account for 59% of global road traffic deaths and 77% of the victims are men.

In a study done by Kanchan et al [15] in Manipal, 89.8% of the victims was 38.7 years. In another study done by Goyal et al [30] in Jaipur, maximum number of cases was in the age group of 21 – 40 years and 87.1% of the

victims were males. Janine jagger et al[45] in a study found that maximum occurrence of head injury occurred in the age group of 20-29 years. The age and gender distribution in our study match well with that of all the other studies.

According to Ministry of Road Transport and Highways (MORTH)[2], India pedestrians, bicyclists and two-wheeler riders comprise the most unprotected road users, accounting for around 40 % of all fatalities. In our study also, we found that pedestrians [80 cases (40%)], pillion riders [19(9.5%)] and drivers of two-wheelers [80 cases (40%)] . In a study by Bayan et al [16] in Pune also, they had found that pedestrians were the most vulnerable group followed by drivers and pillion riders of two-wheelers. Whereas in a study by Kanchan et al [15] in Manipal the most common offending agents were found to be heavy motor vehicles (35.2%) followed by followed by light motor vehicles (31.7%).

When analyzing the contributing factors for the occurrence of road traffic accidents in our study, out of the 99 victims who were drivers or pillion riders of two-wheelers, 80 had not used helmet and hence succumbed to head injuries.

In another study done by Sreedharan et al [17] in Kerala, among the 309 motorcyclist interviewed 78.6% were not wearing helmets. According to WHO[1], wearing helmet can reduce the risk of deaths by almost 40% and the

risk of severe injury by over 70%. Hence strict laws should be enforced to make helmet use mandatory for the drivers of two-wheelers and pillion riders.

In the present study of 200 cases, maximum number of persons sustained fatal head injuries in the day time.

Similar observations were found in the study done by Janine et al[45.]

Preponderance of occurrence of fatal head injuries in road traffic accidents during day time can be explained by the fact that active work is done at day time.

Another common risk factor for RTAs is drunken driving. In a systematic review done by Das A et al [14], 2-33% of the injured and 6-48% of killed RTA victims had consumed alcohol or drugs. In our study however only five victim was reported to have consumed alcohol. This should be because of under reporting and lack of data regarding alcohol use.

Recently there has been a marked increase around the world in the use of mobile phones by drivers that is becoming a growing concern for road safety. But in our study, no data was available regarding the use of mobile phones by the accident victims. Other risk factors like bad illumination, rain, pet or domestic animals and natural diseases contributed very less to the occurrence of RTAs in our study.

Majority of the patients were managed conservatively and only 23 cases (11.6%) had undergone surgery. The period of survival was less than a week in the majority of cases. In approximately 22% of the cases, the period of survival was > 7 days. Moreover according to Dolinskas[39], intracerebral hemorrhages can occur at any time between one to seven days after the infliction of injuries.

The maximum number of deaths occurred during the first 24 hours can be explained by the fact that intracranial haemorrhages,contusions of the brain ,laceration of the brain and edema of the brain which are not compatible with life can occur immediately .

In 16 cases (8%) of the fatal head injury cases death occurred on the spot .

In the present study, among scalp injuries contusion of the scalp was the commonest scalp injury 190 cases as shown in **Table 3**, followed by lacerations 20 cases and abrasions. Frontal, parietal and temporal regions of the scalp were the commonest regions to be involved.

When the fractures of the skull vault are analysed, linear fractures were the commonest type (81) followed by comminuted (8) and depressed (6) fractures as shown in **Table 5**.

This correlates with the study done by Jacobsen et al [23] in Copenhagen, where linear fracture was the commonest type followed by comminuted, depressed, ring and spider web fractures. In the Jaipur study by Goyal et al [30], linear fractures were the commonest followed by depressed and then the comminuted fractures. Considering the predominant site of the skull fractures, frontal and temporal fractures were much more common than parietal and occipital fractures. This is because of the mechanism of most road traffic accidents exposing the fronto-temporal region to risk of trauma than the parieto-occipital region

The incidence of the fractures of the base of skull in the present study was 78 cases which was much higher when compared with the previous studied [Goyal et al [30] (< 1.1%).

In the majority of the cases of fatal head injury ,there was a combination of fracture of the vault of the skull ,intracranial haemorrhages and fracture of the base of the skull.This can be explained by the fact that ,fracture commences at the region of maximum impact and then radiates downwards to the base of the skull.

In the present study base of the Skull was fractured in 86 cases. as shown in Table 17. Fracture of the middle cranial fossa was observed in 51 percent of cases and least in posterior cranial fossa 17%.

In a study of 20 cases of fatal head injury by Devadiga & jain ,12 of these had fracture of both vault and base of the skull. Of the remaining cases 6 had fracture of the base of the skull and 8 cases had fracture of the vault of the skull.

Davidson et al [39] in a study showed combination of fracture of vault of the skull and base of the skull.

Findings in the present study are in correlation of the above findings.

The most common extra axial hemorrhage in our study was subdural 190 cases, followed by subarachnoid169 cases ,whereas in most of the previous studies [Jacobsen et al [31], Bhat VJ et al [32]], subarachnoid hemorrhage was the commonest. . Subdural haemorrhage was observed in 190 Cases. Subdural haemorrhage was the most common intracranial haemorrhage 95% followed by subarachnoid haemorrhage 84.5%, extradural haemorrhage 14% and intracerebral haemorrhage.9.5%

Walpole Levin[34] in a study showed that in fatal head injury cases of road traffic accidents ,Subdural haemorrhage occurred in 60% of the cases.

Reddy [36]found that 53.5% of the intracranial haemorrhages were subdural, 28% were subarachnoid and 25% of the haemorrhages were extradural.

Our observations are in correlation with the above findings.

In the present study ,it is evident that subdural haemorrhages is one of the commonest intracranial haemorrhages followed by subarachnoid haemorrhage, extradural haemorrhage, intraventricular haemorrhage and intracerebral haemorrhage as noted in **Table 11**.

In our study contusions were more common in the frontal and temporal regions than in the parietal and occipital regions, which is similar to the findings of the study by Goyal et al [30]

In the present study of 200 cases of fatal head injuries in road traffic accidents, in addition to other parts of the body were also involved in addition to craniocerebral injury. Chest injury11 (5.5%) cases ,rib fracture 6 cases (3.0%) and fracture of the extremities 8 cases were found.

Walpole Levin [34] found that commonest involvement of chest, face, limbs and ribs in addition to head injury and it correlated very well with our present study.

As the type of vehicle causing the accident is considered there is common involvement of two wheelers followed by four wheelers.

Karrae Solheim [37] found that out of 168 vehicles which were involved in road traffic accidents, lorry, truck and bus were involved in road traffic accidents followed by motor cycles in 6 cases

The findings of the present study collaborates well with the above findings.

CONCLUSION

CONCLUSION

The present study of **"THE STUDY OF PATTERN OF INTRACRANIAL HEMORRHAGE IN FATAL HEAD INJURY CASES IN ROAD TRAFFIC ACCIDENT"** 200 cases were studied which were autopsied.

1. The most vulnerable age group was 31-50 years followed by 21-30 years, 83% of the victims were men..
2. 80% of the victims were pedestrians or two-wheeler riders. Majority of the two-wheeler victims were not wearing helmets at the time of the accident and hence sustained significant head injuries.
3. In majority of the cases (78%), the period of survival was less than a week.
4. The maximum number of accidents causing fatal head injuries were observed during day time 134 cases as compared to night time 66 cases.
5. The motor cyclist and pedestrian were the commonest group of victims in vehicular accidents 160 cases (80%) followed by two wheeler pillion riders.
6. Two wheeler was the commonest offending vehicle followed by four wheeler.
7. Scalp injury with fracture of the skull, fracture of the base of the skull and intracranial hemorrhage was the commonest presentation in fatal head injury cases of road traffic accidents.

8. Totally 96 cases fractures of the vault of the skull were detected in the autopsy. Linear or fissure fractures were the most common type seen in 81 cases, 8 cases were comminuted fractures and 6 cases were depressed fractures.
9. On considering the most common site of fractures in the skull vault, temporal bone was the commonest bone to be fractured followed by frontal bones.
10. Fracture of the base of the skull was observed in 86 cases. The floor of middle cranial fossa was most commonly fractured 51 cases (23.5%) followed by floor of posterior cranial fossa 17 cases (8.5%)
11. Subdural haemorrhage was the most commonest intracranial hemorrhage observed in 190 cases (95.0%) followed by subarachnoid haemorrhage 169 cases (84.5%).Brain stem hemorrhage was found in 13 cases (6.5%)
12. Contusion of the scalp was the commonest scalp injury 190 cases followed by laceration
13. Laceration of the brain parenchyma was found in 20 cases.(10%)
14. In addition to head injury, chest upper limb and lower limbs are commonly involved in road traffic accidents.
15. Two wheeler drivers and pedestrians were more commonly involved in Fatal head injuries in road traffic accidents followed by two wheeler pillion rider.
16. As regard to the type of vehicle ,two wheeler was the common offending vehicle .

RECOMMENDATIONS

RECOMMENDATIONS

1. A national level registry must be established for registering all road traffic accidents all over the country. It will give insight about the epidemiological correlates and risk factors of RTAs, which will help in taking appropriate preventive measures.

2. Modifiable risk factors contributing to the occurrence of RTAs should be brought under control. Use of helmets for two-wheeler riders should be strictly enforced by law. Drunken driving and use of mobile phones while driving should be strictly prohibited. Use of seat belts should be made compulsory. Traffic rules including the lane discipline should be enforced strictly. Bad roads should be repaired and adequate lighting should be provided in all the roads.

3. Emergency contact numbers should be provided in all the roads. Health care facilities should be improved to provide timely interventions to RTA victims. Adequate ambulance facilities should be made available.

4. Standardised national level guidelines should be developed for the management of RTA victims in order to improve their survival.

5. Strict actions should be enforced against the negligent drivers for rash driving.

6. Drivers to be carefully scrutinised before issuing driving license.

7. Traffic rules to be enforced strictly.

8. Use of seat belts and wearing of helmets to be made compulsory for the occupants of vehicle.

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BIBLIOGRAPHY

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ANNEXURE

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301A
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.A. Nirmala.
Post Graduate in MD Forensic Medicine
Institute of Forensic Medicine
Madras Medical College
Chennai 600 003

Dear Dr.A.Nirmala,

The Institutional Ethics Committee has considered your request and approved your study titled **"THE STUDY OF PATTERN OF INTRACRANIAL HAEMORRHAGES IN FATAL HEAD INJURY CASES OF ROAD TRAFFIC ACCIDENT' NO. 15082016.**

The following members of Ethics Committee were present in the meeting hold on **02.08.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.P.Raghumani,MS, Prof. of Surgery,RGGGH,Ch-3	: Member
6.Prof.R.Padmavathy,MD,Director, Inst.of Path,MMC,Ch-3	: Member
7.Prof.S.Tito, MD, Director,Inst.of Int.Med.,MMC,Ch-3	: Member
8.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
9.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
10.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

For DEAN
Madras Medical College
Madras-600 003

URKUND

Urkund Analysis Result

Analysed Document: Nirmala Thesis Final.docx (D31205460)
Submitted: 10/11/2017 8:15:00 AM
Submitted By: nirmalmanju643@gmail.com
Significance: 1 %

Sources included in the report:

ANTO FINAL.doc (D30751905)
Subhas Chandra Debnath_Medicine.pdf (D15260576)

Instances where selected sources appear:

4

CERTIFICATE – II

This is to certify that this dissertation work titled **"THE STUDY OF PATTERN OF INTRACRANIAL HAEMORHAGES IN FATAL HEAD INJURY CASES OF ROAD TRAFFIC ACCIDENT"** of the candidate **Dr. Dr. A.NIRMALA** with Registration Number **201524003** for the award of M.D. in the branch of **FORENSIC MEDICINE.** I personally verified the urkund.com website for plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 1 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

MASTER CHART

SL.NO	PM NO.		AGE	SEX	OCCUPATION	MODE OF ACCIDENT	TIME	DRIVER/OCCUPANT/PEDESTRAN	HOSPITAL	NUMBER OF STAY IN HOSPITAL	SURGERY	SDH	EDH	SAH	ICH	IVH	FRACTURE	SCALP CONTUSION	BASE OF SKULL	CONTRIBUTORY FACTORS	OTHER INJURIES	LACERATION	BRAIN
1	1902/16	RANGAN	40	MALE	COOLIE	TWO WHELER VS LORRY	2.00 PM	DRIVER	YES	7DAYS	NO	NIL	NO	NIL	NO	NO	DEPRESSED FRACTURE	RT FTP	INTACT	NO	CHEST INJURY	NIL	NORMAL
2	1903/16	MANI	45	MALE	WATCMAN	PEDESTRAN VS TWO WHEELER	4.30PM	PEDESTRAN	YES	1DAY	NO	PRESENT AII OVER	NO	LT FTP	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	RT FTP	INTACT	NO	RIB FRACTURE	NIL	NORMAL
3	1904/16	PANDURANGAN	52	MALE	DRIVER	ACCIDENTAL FALL FROM TWO WHEELER	8.20AM	DRIVER	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	COMMINUTED FRACTURE	BOTH FTP	COMMINUTED FRACTURE	NO	NO	IL	NORMAL
4	1906/16	RAJKUMAR	35	MALE	COOLIE	SELF FALL FROM TWO WHEELR	10.00PM	DRIVER	BROUGHT DEAD	NA	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	COMMINUTED FRACTURE	BOTH FTP	COMMINUTED FRACTURE	YES(ALCOHOL)	NO	LACERATION LT FRONTOPARIETAL	OEDEMATOUS
5	1906/16	ELIYAS BASHA	20	MALE	COOLIE	TWO WHEELER VS TWO WHEELER	4.30AM	DRIVER	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	BOTH FTP	INTACT	YES(ALCOHOL)	FRACTURE NASAL BONES,FRACTURE RADIUS AND ULNA	NIL	NORMAL
6	1909/16	Y.NIRMALA	42	FEMALE	HOUSE WIFE	FALL from TWO WHEELER	8.30AM	PILLON	YES	1 DAY	NO	RIGHT FTP	NO	DIFFUSE	NO	NO	INTACT	BOTH FTP	MCF AND PCF	NO	NO	NIL	NORMAL
7	1910/16	SHANTHI	38	FEMALE	HOUSE WIFE	PEDESTRAN VS TWO WHEELER	8.30 PM	PEDESTRAN	YES	4 DAY	YES	LEFT FTP	NO	NO	NO	NO	INTACT	LT FTP	RT MCF	NO	NO	NIL	NORMAL
8	1911/16	BOOPALAN	45	MALE	COOLIE	TWO WHEELR VS TWO WHEELER	2.30 PM	DRIVER	YES	ONE DAY	NO	RIGHT FTP	NO	RIGHT FTP	NO	NO	FISSUR FRACTURE RT PARIETAL AND TEMPORAL BONE	RT FTP	INTACT	NO	NO	NIL	OEDEMATOUS
9	1912/16	DHANALAKSHMI	65	FEMALE	HOUSEWIFE	PEDESTRAN VS TWO WHEELER	2.30 PM	PEDESTRAN	YES	ONE DAY	NO	RIGHT FTP	NO	RIGHT FTP	NO	NO	FISSURE FRACTURE RIGHT PARIETAL AND TEMPORAL BONE	RT FTP	INTACT	NO	NO	NIL	OEDEMATOUS
10	1913/16	VANATHATCHI	51	FEMALE	HOUSEWIFE	PEDESTRAN VS TWO WHEELER	8.00AM	PEDESTRAN	YES	7 DAYS	NO	DIFFUSE FTP	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LEFT TEMPORAL BONE AND PARIETAL	LT FTP	LEFT MCF	NO	NO	NIL	OEDEMATOUS
11	1914/16	SELVAKUMAR	55	MALE	DRIVER	CAR VS LORRY	5.25PM	DRIVER	YES	ONE DAY	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	HINGE FRACTURE	LT FTP	RT MCF AND RT LT MCF	NO	NO	NIL	OEDEMATOUS
12	1918/16	LALU SHARMA	52	MALE	CARPENTER	PEDESTRAN VS TWO WHEELER	7.15 AM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	NO	PRESENT	NO	FISSURE FRACTURE OCCIPITAL BONE	DIFFUSE	RT MCF	NO	NO	NIL	NORMAL
13	1920/16	G.BOOPATHY	52	MALE	PLUMBER	PEDESTRAN VS TWO WHEELER	5.00AM	PEDESTRAN	NO	NO	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE FRACTURE RT FRONTAL BONE	RT FTP	PCF	NO	NO	NIL	NORMAL
14	1925/16	UNKNOWN	45	MALE	BEGGAR	PEDESTRAN VS TWO WHEELER	8.00PM	PEDESTRAN	NO	NO	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE FRACTURE RT TEMPORAL	RT FTP	RT PCF	NO	NO	NIL	OEDEMATOUS
15	1926/16	VARADHAN	50	MALE	OFFICE GOER	PEDESTRAN VS TWO WHEELER	7.15PM	PEDESTRAN	YES	ONE DAY	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	NO	NIL	OEDEMATOUS
16	1928/16	ELUIMALAI	50	MALE	COOLIE	PEDESTRAN VS TWO WHEELER	5.50PM	PEDESTRAN	YES	TWO	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	NO	PCF	NO	NO	NIL	NORMAL
17	1931/16	KESAVAN	50	MALE	COOLIE	TWO WHEELER VS FOUR WHEELER	5.50PM	TWO WHEELER	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NO	INTACT	RT FTP	INTACT	NO	NO	NIL	NORMAL
18	1932/16	KANNIGA	68	FEMALE	HOUSE WIFE	SELF FALL FROM TWO WHEELR	8.15 PM	PILLON RIDER	YES	6 DAYS	YES	LEFT FTP	NO	NO	NIL	NO	INTACT	DIFFUSE	INTACT	NO	NO	NIL	OEDEMATOUS
19	1933/16	MARIYAPPAN	34	MALE	DRIVER	UNKNOWN VEHICLE	2.30 PM	PEDESTRAN	YES	ONE DAY	NO	DIFFUSE	NO	DIFFUSE	NIL	NIL	FISSURE FRACTURE RIGHT TEMPORAL BONE	RT FTP	INTACT	NO	NO	NIL	NORMAL
20	1934/16	IRUDAYA RAJ	50	MALE	PEDESTRAN	UNKNOWN VEHICLE	8.15PM	PEDESTRAN	YES	TWO DAYS	NO	DIFFUSE	NO	DIFFUSE	NIL	NIL	INTACT	NO	INTACT	NO	NO	NIL	NORMAL
21	1944/16	MAAVAN	35	MALE	TWO WHEELER RIDER	TWO WHEELER VS FOUR WHEELER	5.15 PM	TWO WHEELER RIDER	YES	6 DAYS	YES	DIFFUSE	NO	DIFFUSE	NIL	NIL	INTACT	RT FTP	RT MCF	NO	NO	NIL	NORMAL
22	1945/16	M.YAMUNA	44	FEMALE	COOLIE	SELF FALL FROM TWO WHEELR	7.30 PM	PILLION RIDER	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NIL	INTACT	RT PARIETAL	INTACT	NO	RIB FRACTURE	NIL	NORMAL
23	1946/16	MOHAMMAD ISMAIL	38	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	10.30 PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NIL	INTACT	RT FTP	LEFT PCF	NO	NO	NIL	NORMAL
24	1953/16	SATISH	33	MALE	CAR DRIVER	TWO WHEELER VS TEMPO TRAVELLER	11.15 PM	TWO WHEELER RIDER	YES	3 DAYS	NO	DIFFUSE	NO	DIFFUSE	NIL	NIL	INTACT	MID PARIETAL	LT PCF	NO	NO	LACERATION RT FRONTOTEMPORAL	NORMAL
25	1954/16	RAJENDRAN	68	MALE	CAR DRIVER	TWO WHEELER VS TWO WHEELER	5.30 PM	TWO WHEELER RIDER	SPOT DEAD	NO	NO	RIGHT PARIETAL	NO		NIL	NIL	INTACT	RT PARIETAL	INTACT	NO	NO	LACERATION RT FRONTOTEMPORAL	NORMAL
26	1955/16	THIRUMALAI	32	MALE	COOLIE	TWO WHEELER VS TWO WHEELER	6.30 PM	TWO WHEELER RIDER	YES	1 DAY	NO	DIFFUSE RIGHT	RT PARIETAL	DIFFUSE	PRESENT	NIL	FISSURE FRACTURE RT PARIETAL BONE	RT FTP	RT MCF	ALCOHOL	NO	NIL	OEDEMATOUS
27	869/17	KARUPPAN	85	male	RETIRED	SELF FALL FROM FROM TWO WHEELER	8.30AM	PILLION RIDER	BROUGHT DEAD	NO	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NIL	FISSURE FRACTURE LT TEMPORAL BONE	LT FTP	INTACT	NIL	RIB FRACTURE	NIL	NORMAL
28	855/17	AAMIN MUTHU	60	FEMALE	UNEMPLOYED	PEDESTRAN VS FOUR WHEELER	11 00AM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	RT FTP	INTACT	NO	NO	NIL	NORMAL
29	853/17	PARVATHY MUTHU	67	FEMALE	HOUSE WIFE	SELF FALL FROM FROM TWO WHEELER	11.00 AM	PILLION RIDER	YES	7 DAYS	YES	PO	NO	BOTH	NO	NIL	BONY AND DURAL DEFECT BOTH FRONTAL REGIONS	DIFFUSE	INTACT	NO	NO	BILATERAL FRONTAL CONTUSION	OEDEMATOUS
30	846/17	KUMAR	35	MALE	SELF EMPLOYED	TWO WHEELER VS FOUR WHEELER	2.00PM	DRIVER OF TWO WHEELER	BROUGHT DEAD	NIL	NO	DIFFUSE	NO	NO	NO	NIL	INTACT	DIFFUSE	NIL	NO	FRACTURE LEFT TIBIA,FIBULA AND FEMUR	NIL	NORMAL
31	1956/17	MUTHUSELVAN	51	male	COOLIE	FALL FROM GOVT BUS	7.30 PM	PASSENGER	yes	7 days	NO	diffuse	NO	DIFFUSE	PRESENT	nil	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF TO LT SIDE	YES(ALCOHOL)	NO	NIL	OEDEMATOUS
32	1957/17	RAMANATHAN	61	MALE	MECHANIC	PEDESTRAN VS TWO WHEELER	8.20 PM	PEDESTRAN	YES	ONE DAY	NO	RIGHT FTP	NO	DIFFUSE	NO	NIL	FISSURE FRACTURE RT TEMPORAL BONE	LT FTP	INTACT	NO	NO	NIL	NORMAL
33	1964/17	MAHESH	35	MALE	COOLIE	PEDESTRAN VS TWO WHEELER	5.30PM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	PARIETAL BONE	NO	INTACT	NO	NO	NIL	NORMAL
34	1970/17	VEL	42	MALE	COOLIE	BICYCLIST VS TWO WHEELER	11.00 AM	CYCLIST	YES	4DAYS	YES	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	RT CRANIOTOMY 6 BURR HOLES	RT FTP	RT MCF	NO	CHEST INJURY	NIL	NORMAL
35	1973/17	ARJUN	26	MALE	COOLIE	SELF FALL FROM CYCLE	2.30 PM	CYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	FISSURE FRACTURE LT OCCIPITAL BONE	RT FTP	RT PCF	NO	NO	NIL	NORMAL
36	1981/17	SANKAR	21	MALE	COOLIE	PEDESRRA VS FOUR WHEELER	7.30PM	PEDESTRAN	YES	3DAYS	NO	DIFFUDE	RT TEMPORAL	DIFFUSE	NO	NIL	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	NO	NIL	NORMAL
37	1985/17	PRAVEEN	21	MALE	COOLIE	PEDESTRAN VS TWO WHEELER	4.40 AM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	NO	INTACT	NO	NO	NIL	NORMAL
38	1988/17	REVATHY	28	FEMALE	COOLIE	SELF FALL FROM TWO WHEELR	10.30 AM	WORKER	YES	4DAYS	NO	RT FTP	NO	RT FTP	NO	NIL	HINGE FRACTURE	BOTH PARIETO	INTACT	NO	NO	NIL	NORMAL
39	1991/17	RISHI	43	MALE	SECURITY	PEDESTRAN VS TWO WHEELER	8.40 AM	PEDESTRAN	YES	2 DAYS	NO	LT FTP	NO	LT FTP	NO	NIL	FISSURE FRACTURE OCCIPITAL BONE	RT FTP	INTACT	NO	NO	NIL	NORMAL
40	2020/16	RAMACHANDRAN	55	MALE	SECURITY	PEDESTRAN VS TWO WHEELER	7.00 AM	PEDESTRAN	YES	1 DAY	YES	LT FTP	NO	NO	NO	NIL	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	INTACT	NO	NO	NIL	OEDEMATOUS
41	2019/16	SENTHIL	34	MALE	COOLIE	PEDESTRAN VS UNKNOWN VEHICLE	7.00 AM	PEDESTRAN	YES	8 DAY	YES	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	DIFFUSE	INTACT	ALCOHOL	LIVER LACERATION	NIL	NORMAL
42	2015/16	VIMAL RAJ	27	MALE	DRIVER	TWO WHEELR VS FOUR WHEELER	12.00 AM	MOTORCYCLIST	YES	14 DAYS	YES	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NIL	NORMAL
43	2029/16	KUMAR	39	MALE	COOLIE	SELF FALL FROM TWO WHEELR	12.45PM	MTORCYCLIST	YES	3 DAYS	NO	DIFFUSE BOTH CEREBRAL	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT OCCIPITAL BONE	LEFT FTP	LT ACF	NO	NO	NIL	NORMAL
44	2031/16	VELAYUDHAM	32	MALE	DRIVER	SELF FALL FROM TWO WHEELR	1.00PM	MOTORCYCLIST	YES	4DAYS	NO	DIFFUSE BOTH CEREBRAL	NO	DIFFUSE	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NIL	NORMAL
45	2032/16	C1 SRIPERAMBUDUR	36	MALE	PAINTER	TWO WHEELER VS CAR	5.00 PM	MOTORCYCLIST	YES	3 DAYS	YES	CEREBRAL AND CEREBELLAR	RT FP	CEREBRAL AND CEREBELLAR	NO	NO	FISSURE FRACTURE LT PARIETAL BONE AND TEMPORAL BONE	RT FTP	LT MCF	NO	NO	NIL	NORMAL
46	2041/16	THIRUKOVILOOR	47	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	3.15 PM	PEDESTRAN	YES	4 DAYS	NO	CEREBRAL AND CEREBELLAR	NO	CEREBRAL AND CEREBELLAR	NO	NIL	INTACT	DIFFUSE	INTACT	NO	NO	NIL	NORMAL
47	2045/16	RAJA	20	MALE	DRIVER	TWO WHEELER VS VAN	5.30 PM	MOTORCYCLIST	YES	3 DAYS	YES	CEREBRAL AND CEREBELLAR	NO	CEREBRAL AND CEREBELLAR	NO	NIL	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NIL	NORMAL
48	926/16	RAMESH	35	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELR	9.30 PM	PILLION RIDER	YES	3DAYS	NO	CEREBRAL BOTH SIDES	NO	CEREBRAL	NO	NIL	INTACT	LEFT FTP	INTACT	NO	NO	NIL	NORMAL
49	928/16	DEVENDRA KUMAR	50	MALE	COOLIE	TWO WHEELER VS UNKNOWN VEHCLE	4.30 PM	TWO WHEELER RIDER	YES	1 DAY	NO	BOTH CEREBRAL	NO	BOTH CEREBRAL	NO	NIL	INTACT	DIFFUSE	INTACT	NO	NO	NIL	NORMAL

50	929/16	KUPPUSWAMY	25	MALE	DRIVER	SELF FALL FROM TWO WHEELR	4.30 PM	SELF FROM TWO WHEELER	YES	1 DAY	NO	DIFFUSE	RT TP	DIFFUSE	NO	NIL	FISSURE FRACTURE LT OCCIPITAL BONE	DIFFUSE	RT MCF AND RT ACF	NO	NO	NIL	OEDEMATOUS
51	930/16	KESAVAN	51	MALE	COOLIE	SELF FALL FROM TWO WHEELR	4.00PM	SELF FROM TWO WHEELER	YES	4 DAYS	NO	BOTH CEREBRAL	NO	BOTH CEREBRAL	NO	NIL	FISSURE FRACTURE RT TEMPORAL BONE	BOTH FTP	LT MCF	NO	NO	NIL	NORMAL
52	931/16	PITCHAI MUTHU	34	MALE	SELF EMPLOYED	TWO WHEELER VS FOUR WHEELER	245 PM	TWO WHEELER RIDER	YES	1 DAY	YES	DIFFUSE	NO	DIFFUSE	NO	NIL	BONY AND DURAL DEFECT LEFT FTP	BOTH FTP	INTACT	NO	NO	NIL	NORMAL
53	932/16	DEVENDRAN	58	MALE	COOLIE	BICYCLE VS BUS	9.05 AM	CYCLIST	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	DIFFUSE	INTACT	NO	NO	NIL	NORMAL
54	935/16	JANAKI	43	MALE	HOUSE WIFE	ACCIDENTAL FALL FROM TWO WHEELER	4.30 PM	PILLION RIDER	YES	1 DAY	NO	LT FTP	NO	LT FTP	NO	NIL	FISSURE FRACTURE OCCIPITAL BONE	RT FTP	RT PCF	NO	NO	LACERATION LT TEMPORAL	OEDEMATOUS
55	950/16	SUNDAR RAJ	35	MALE	DRIVER	DETAILS NK	2.30 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	LEFT PARIETAL	DIFFUSE	NO	NIL	FISSURE FRACTURE RT PARIETAL BONE	LT FP	RT AND LT MCF	NO	NO	NIL	NORMAL
56	954/16	VADIVEL	56	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELR	3.00 PM	MOTORCYCLIST	YES	1 DAY	NO	LT CEREBRAL	LEFT CEREBRAL	LT FTP	NO	NIL	INTACT	NO	INTACT	NO	NO	NIL	OEDEMATOUS
57	953/16	SATHISH	25	MALE	COOLIE	TWO WHEELER VS TWO WHEELER	9.45 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NIL	FISSURE FRACTURE RT PARIETAL BONE	LT FTP	RT AND LT MCF	NO	NO	NIL	OEDEMATOUS
58	968/26	CHINNATHAI	60	FEMALE	HOUSE WIFE	FALL FROM GOVT BUS	4.35 PM	PASSENGER	YES	25 DAYS	NO	LT CEREBRAL	NO	LT CEREBRAL	NO	NIL	INTACT	LT FTP	INTACT	NO	NO	NIL	NORMAL
59	969/16	LOGAMMAL	66	FEMALE	HOUSE WIFE	PEDESTRIAN VS TWO WHEELER	7.30 PM	PEDESTRAN	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NIL	INTACT	BOTH FTP	INTACT	NO	NO	LACERATION RT FRONTOTEMPORAL	OEDEMATOUS
60	970/16	MUTHUSELVAN	43	MALE	COOLIE	PEDESTRIAN VS CAR	2.20 PM	PEDESTRAN	YES	3 DAYS	NO	NIL	NO	DIFFUSE	NO	YES	FISSURE FRACTURE RT PARIETAL BONE	RT FTP	COMMINUTED FRACTURE	NO	NO	NIL	OEDEMATOUS
61	971/16	GUNASEKAR	60	MALE	DRIVER	PEDESTRIAN VS TWO WHEELER	9.15 PM	PEDESTRAN	YES	10 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	RT PT	INTACT	NO	NO	NIL	NORMAL
62	974/16	ANNAMMAL	50	FEMALE	HOUSE WIFE	PEDESTRIAN VS CAR	4.45 PM	PEDESTRAN	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	NO	INTACT	NO	NO	NIL	NORMAL
63	975/16	RADHA	45	FEMALE	HOUSE WIFE	PEDESTRIAN VS CAR	6.25 PM	PEDESTRAN	BROUGHT DEAD	NIL	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE LT TEMPORAL BONE	LT FTP	LT MCF	NO	NO	NIL	OEDEMATOUS
64	976/16	MADHANSHARMA	35	MALE	DRIVER	SELF FALL FROM TWO WHEELR	6.00 PM	MOTORCYCLIST	YES	17 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	DEPRESSED FRACTURE RT PARIETAL BONE	LT FTP	INTACT	NO	NO	NIL	NORMAL
65	978/16	RAJAVEL	23	MALE	SELF EMPLOYED	CYCLE VS AUTO	7.30 PM	CYCLIST	YES	1 DAY	NO	LT FTP	NO	LT FTP	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	RT FTP	LT ACF	NO	NO	NIL	NORMAL
66	981/16	MUNEER AHMED	28	MALE	SELF EMPLOYED	TWO WHEELER VS FOUR WHEELER	11.15 PM	MOTORCYCLIST	YES	2 DAYS	NO	DIFFUSE	NO	NO	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	NO	LACERATION RT FRONTAL	OEDEMATOUS
67	983/16	SURYA PRAKASH	50	MALE	PLUMBER	TWO WHEELER VS CAR	2.05 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	LT FTP	INTACT	NO	NO	NO	NORMAL
68	985/16	SENTHIL KUMAR	27	MALE	PAINTER	PEDESTRIAN VS FOUR WHEELER	7.00 PM	PEDESTRAN	BROUGHT DEAD	NIL	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	NO	INTACT	NO	NO	NO	NORMAL
69	988/16	UNKNOWN	35	MALE	UNKNOWN	PEDESTRIAN VS UNKNOWN VEHICLE	7.55PM	PEDESTRAN	YES	4 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	INTACT	NO	INTACT	NO	NO	NO	OEDEMATOUS
70	989/16	BALA RAJESHWARAN	30	MALE	DRIVER	PEDESTRIAN VS TWO WHEELER	6.OO PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	YES	FISSURE FRACTURE OCCIPITAL BONE	OCCIPITAL	LT PCF	NO	NO	NO	OEDEMATOUS
71	991/16	MURUGAN	52	MALE	SELF EMPLOYED	PEDESTRAIN VS TWO WHEELER	12.40 PM	PEDESTRAN	YES	5 DAYS	YES	DIFFUSE	RT PARIETAL	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	LT FTP	INTACT	NO	NO	NO	OEDEMATOUS
72	992/16	VIMAL	68	MALE	COOLIE	SELF FALL FROM TWO WHEELER	1.00 PM	MOTORCYCLIST	YES	8 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	DIFFUSE	LEFT MCF	NO	NO	NO	OEDEMATOUS
73	993/16	RAMAKRISHNAN	84	MALE	DRIVER	PEDESTRIAN VS TWO WHEELER	5.35 PM	PEDESTRAN	YES	1 DAY	NO	DIFUSE	NO	DIFFUSE	NO	NO	LT FRONTO PARIETAL BONE	LT FTP	RIGHT MCF	NO	NO	NO	OEDEMATOUS
74	995/16	BABU	25	MALE	COOLIE	SELF FALL FROM TWO WHEELR	11.45 PM	MOTORCYCLIST	YES	6 DAYS	YES	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT PARIETAL BONE	RT FTP	RIGHT MCF	NO	NO	NO	OEDEMATOUS
75	997/16	MARIMUTHU	33	MALE	PAINTER	DETAILS NK	9.45 PM	MOTORCYCLIST	YES	1 DAY	NO	BOTH FRONTAL AND TEMPORAL	NO	BOTH FRONTAL AND TEMPORAL NO		NO	FISSURE FRACTURE OCCIPITAL BONE	LT FTP	PCF	NO	NO	NO	OEDEMATOUS
76	998/16	DAMODARAN	68	MALE	COOLIE	PEDESTRIAN VS TWO WHEELER	7.35 PM	PEDESTRAN	YES	13 DAYS	NO	DIFFUSE	NO	NO	NO	DIFFUSE	INTACT	NO	INTACT	NO	NO	NO	NORMAL
77	999/16	UNKNOWN	70	MALE	BEGGAR	PEDESTRAN VS TWO WHEELER	5.45 PM	PEDESTRAN	YES	6 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	LEFT PCF	NO	NO	BILATERAL FRONTAL CONTUSION	NORMAL
78	1000/16	ANDAL	45	FEMALE	HOUSEWIFE	PEDESTRAN VS TWO WHEELER	9.00 PM	PEDESTRAN	YES	1 DAY	NO	LT FTP	NO	NO	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
79	1003/16	ARUMUGAM	46	MALE	DRIVER	TWO WHEELER VS TWO WHEELER	9.30 PM	MOTORCYCLIST	YES	6 DAYS	NO	LT CEREBRAL	NO	LT CEREBRAL	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
80	1004/16	MAHESWARI	23	FEMALE	HOUSE WIFE	PEDESTRAN VS CAR	6.00 AM	PEDESTRAN	BROUGHT DEAD		NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
81	1015/16	GIRI	37	MALE	DRIVER	UNKNOWN VEHICLE	7.00 PM	PEDESTRAN	YES	2 DAYS	NO	LEFT FTP	NO	LT FTP	NO	NO	BONY AND DURAL DEFECT	LT FTP	RT MCF	LT PARIETAL	NO	NO	OEDEMATOUS
82	1017/16	SELVARAJ	55	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	9.45PM	PEDESTRAN VS TWO WHEELER	YES	1 DAY	NO	LEFT CEREBRAL	NO	LEFT CEREBRAL	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
83	1018/16	ESWARIAH	30	MALE	COOLIE	PEDESRRA VS TWO WHEELER	12.45 PM	PEDESTRAN VS TWO WHEELER	YES	1 DAY	NO	LEFT CEREBRAL	NO	LEFT CEREBRAL	NO	NO	INTACT	LT CEREBRAL	INTACT	NO	NO	NO	NORMAL
84	1023/16	SHANMUGAM	40	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELR	11.20 PM	MOTORCYCLIST	YES	1 DAY	NO	BOTH PARIETO TEMPORAL	NO	BOTH PARIETO TEMPORAL	NO	PRESENT	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
85	1024/16	MALAR	27	FEMALE	HOUSE WIFE	TWO WHEELER VS TWO WHEELER	6.30 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
86	1025/16	ARUMUGAM	60	MALE	SELF EMPLOYED	PEDESTRIAN VS TWO WHEELER	11.15 AM	PEDESTRAN	YES	1 DAY	NO	RT FTP	NO	NO	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
87	1030/16	SAMIKANNU	60	MALE	COOLIE	CYCLE VS FOUR WHEELER	6.30 PM	CYCLIST	YES	1 DAY	NO	DIFFUSE	OCCIPITAL	DIFFUSE	NO	NO	FISSURE FRACTURE OCCIPITAL BONE	DIFFUSE	INTACT	NO	NO	NO	NORMAL
88	1031/16	MANICKAM	45	MALE	DRIVER	TWO WHEELER VS FOUR WHEELER	1.05 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
89	1036/16	VENKETESAN	47	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELR	11.30 AM	MOTORCYCLIST	YES	1 DAY	NO	LT FTP	NO	LT FTP	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
90	1037/16	SUGUNA	43	FEMALE	COOLIE	PEDESTRAN VS TWO WHEELER	9.30 PM	PEDESTRN	YES	7 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
91	1038/17	SIRANJEEVI	23	MALE	COOLIE	PEDESTRIAN VS TWO WHEELER	6.35 PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
92	1039/16	BASHHEER	50	MALE	PAINTER	TWO WHEELER VS LORRY	7.00 AM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	COMMINUTED FRACTURE VAULT OF SKULL	DIFFUSE	COMMINUTED FRACTURE	NO	NO	NO	NORMAL
93	1040/16	KANDAN	30	MALE	DRIVER	SELF FALL FROM TWO WHEELR	8.10 AM	MOTORCYCLIST	YES	1 DAY	NO	LT FTP	NO	LT FTP	NO	NO	INTACT	NO	INTACT	NO	CERVICAL SPINE	NO	NORMAL
94	1043/16	KALYANI	60	FEMALE	VENDOR	SELF FALL FROM TWO WHEELR	12.25 AM	MOTORCYCLIST	BROUGHT DEAD	NA	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	CHEST INJURY	NO	NORMAL
95	1044/16	VEERAMANI	35	MALE	DRIVER	PEDESTRAN VS TWO WHEELER	8.50 PM	PEDESTRAN	YES	4 DAYS	YES	RT FTP	RT PARIETAL	RT FTP	NO	NO	INTACT	LT FTP	INTACT	NO	CHEST INJURY	NO	NORMAL
96	1045/16	RAVI KUMAR	22	MALE	VENDOR	SELF FALL FROM TWO WHEELR	12.00 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RIGHT FTP	INTACT	NO	INJURY TO RT LOWER LIMB	NO	NORMAL
97	1046/16	NAGARAJ	40	MALE	COOLIE	SELF FALL FROM TWO WHEELR	10.00 AM	MOTORCYCLIST	YES	1 DAY	NO	D	RT PARIETAL	DIFFUSE	PRESENT	NO	COMMINUTED FRACTURE VAULT OF SKULL	DIFFUSE	COMMINUTED FRACTURE	NO	CHEST INJURY	NO	OEDEMATOUS
98	1047/16	DEEPAK	22	MALE	DRIVER	SELF FALL FROM TWO WHEELR	3.00 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
99	1048/16LINGAM		55	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	4.50 PM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
100	1049/16	MANIKANDAN	27	MALE	DRIVER	TWO WHEELER VS GOAT	4.35 PM	MOTORCYCLIST	YES	7 DAYS	NO	LT FTP	NO	LT FTP	NO	NO	DEPRESSED FRACTURE LT FRONTAL BONE	LT FTP	INTACT	NO	NO	NO	NORMAL
101	1050/16	STALIN	27	MALE	SELF EMPLOYED	ACCIDENTAL FALL FROM TWO WHEELER	6.00 PM	MOTORCYCLIST	YES	30 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFFUSE	INTACT	NO	NO	NO	NORMAL
102	1056/16	FRANKLIN	37	MALE	DRIVER	ACCIDENTAL FALL FROM TWO WHEELER	3.00 PM	MOTORCYCLIST	YES	2 DAYS	NO	LT FTP	LT PARIETAL	LT FTP	NO	NO	FISSURE FRACTURE LT PARIETAL BONE	LT FTP	INTACT	NO	NO	NO	NORMAL

103	1057/16	NAGAMANI	56	FEMALE	STAFF	DETAILS NK	4.30 PM	NK	BROUGHT DEAD	NO	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
104	1058/16	VIGNESH	34	MALE	COOLIE	DETAILS NK	1.05 PM	NK	YES	2 DAYS	NO	RT FTP	RT TEMPORAL	RT FTP	PRESENT	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	CHEST INJURY	NO	OEDEMATOUS
105	1062/16	NATRAJ	35	MALE	PAINTER	TWO WHEELER VS TEMPO TRAVELLER	4.00 PM	MOTORCYCLIST	YES	2 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	DEPRESSED FRACTURE RT PARIETAL BONE	DIFFUSE	RT MCF	NO	NO	NO	NORMAL
106	1066/16	RAJ KUMAR	30	MALE	TEACHER	SELF FALL FROM TWO WHEELR	3.30 PM	MOTORCYCLIST	YES	5 DAYS	NO	LT FTP	LT PARIETAL	LT FTP	NO	NO	FISSURE FRACTURE LEFT PARIETAL AND TEMPORAL BONE	DIFFUSE	LT MCF	NO	NO	NO	OEDEMATOUS
107	1068/16	ASHOK KUMAR	33	MALE	COOLIE	TWO WHEELER VS TWO WHEELER	2.00 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	RT TEMPORAL	DIFFUSE	PRESENT	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NO	NORMAL
108	1070/16	DHARANI	48	FEMALE	HOUSEMAID	SELF FALL FROM TWO WHEELR	7.30 PM	PILION RIDER	YES	NO	NO	DIFFUSE	RT TEMPORAL	DIFFUSE	NO	YES	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	INJURY TO RT LOWER LIMB	NO	OEDEMATOUS
109	1071/16	GOVINDASWAMY	60	MALE	COOLIE	TWO WHEELER VS SHARE AUTO	4.45 PM	MOTORCYCLIST	YES	21 DAYS	NO	LEFT FTP	NO	LT FTP	PRESENT	NO	FISSURE FRACTURE LT PARIETAL BONE	DIFFUSE	LT MCF	NO	NO	NO	NORMAL
110	1074/16	SUBRAMANI	64	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	6.05 PM	PEDESTRAN	YES	6 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	DEPRESSED FRACTURE RT PARIETAL BONE	DIFFUSE	RT AND LT MCF	NO	INJURY TO RT UPPER LIMB	NO	OEDEMATOUS
111	1076/16	LAWRENCE	23	MALE	PAINTER	PEDESTRAN VS TWO WHEELER	5.40 PM	PEDESTRAN	BROUGHT DEAD	NA	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	COMMINUTED FRACTURE OF VAULT OF SKULL	DIFFUSE	COMMINUTED FRACTURE OF FLOOR	NO	CHEST INJURY AND INJURY TO LOWER LIMB	NO	OEDEMATOUS
112	1077/16	KARUNANIDHI	53	MALE	COOK	PEDESTRAN VS TWO WHEELER	7.45 PM	PEDESTRAN	BROUGHT DEAD	NA	NO	DIFFUSE	NO	DIFFUSE	PRESENT	PRESENT	HINGE FRACTURE	DIFFUSE	FRACTURE RT MCF	NO	NO	NO	NORMAL
113	1079/16	KARTHIKEYAN	54	MALE	DRIVER	TWO WHEELER VS TWO WHEELER	2.35 PM	MOTORCYCLIST	YES	2 DAYS	NO	LEFT FTP	NO	LEFT CEREBRAL	NO	NO	FISSURE FRACTURE LEFT TEMPORAL BONE	LEFT FTP	FRACTURE LT MCF	NO	NO	NO	NORMAL
114	1080/16	RAJ KUMAR	44	MALE	COOLIE	SELF FALL FROM TWO WHEELR	3.30 PM	MOTORCYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE FRACTURE RT PARIETAL BONE	DIFFUSE	INTACT	NO	NO	NO	NORMAL
115	1081/16	JAYAKUMAR	18	MALE	DRIVER	TWO WHEELER VS TWO WHEELER	10.00 AM	MOTORCYCLIST	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
116	1082/16	ARIFFA	22	MALE	COOLIE	PEDESTRSAN VS TWO WHEELER	10.00 AM	PEDESTRAN	YES	1 DAY	NO	RT FTP	NO	RT FTP	NO	NO	INTACT	RT FTP	INTACT	NO	INJURY TO LT LOWER LIMB	NO	NORMAL
117	1085/16	KANNAN	50	MALE	TEACHER	BICYCLE VS TWO WHEELER	3.00 PM	CYCLIST	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NO	NORMAL
118	1086/16	VIJAY KUMAR	43	MALE	SELF EMPLOYED	ACCIDENTAL FALL FROM TWO WHEELER	7.45 PM	MOTORCYCLIST	YES	2 DAYS	NO	LT FTP	NO	NO	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	LT FTP	LT MCF	NO	NO	NO	NORMAL
119	Jan-16	AYYANAR	16	MALE	COOLIE	TWO WHEELER VS FOUR WHEELER	9.30 PM	TWO WHEELER PILION RIDER	YES	3 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT PARIETAL BONE AND TEMPORAL	DIFFUSE	RT MCF	NO	NO	NO	NORMAL
120	Feb-16	SUMATHI	40	FEMALE	HOUSE WIFE	SELF FALL FROM TWO WHEELR	4.00 PM	TWO WHEELER PILLION RIDER	YES	7 DAYS	NO	RT FTP	RT PARIETAL	RT FTP	NO	PRESENT	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
121	Mar-16	MOORTHI	60	MALE	COOLIE	SELF FALL FROM TWO WHEELR	10.30 AM	MOTORCYCLIST	YES	7DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NO	OEDEMATOUS
122	Oct-16	BABU	35	MALE	DRIVER	TWO WHEELER VS BOLERO	11.30 AM	PEDESTRAN	YES	2DAYS	NO	LT FTP	NO	NO	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	LT FTP	LT MCF	NO	NO	NO	NORMAL
123	Dec-16	PRAKASH	32	MALE	COOLIE	SELF FALL FROM TWO WHEELR	8.30 PM	TWO WHEELER PILLION RIDER	YES	15 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
124	15/16	SIVASANKAR	38	MALE	COOLIE	TWO WHEELER VS FOUR WHEELER	2.00 PM	TWO WHEELER DRIVER	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
125	13/16	KAMALAMMAL	70	FEMALE	SELF EMPLOYED	TWO WHEELER VS TWO WHEELER	1.15 PM	TWO WHEELER DRIVER	YES	2 DAYS	NO	LT FTP	NO	NO	NO	NO	FISSURE FRACTURE LT PARIETAL BONE	DIFFUSE	INTACT	NO	NO	NO	NORMAL
126	16/16	THIRUMALAI	55	MALE	SELF EMPLOYED	TWO WHEELR VS FOUR WHEELER	4.00 PM	TWO WHEELER DRIVER	YES	4 DAY	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	INTACT	NO	NO	NO	NORMAL
127	17/16	KUMARAVEL	34	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELR	6.00 PM	TWO WHEELER PILLION RIDER	YES	6 DAYS	NO	DIFFUSE	RT PARIETAL	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
128	20/16	DHANALAPAL	45	MALE	GARDENER	TWO WHEELER VS HEAVY VEHICLE	9.45 PM	TWO WHEELER DRIVER	YES	2 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	LACERATION RT TEMPORAL LOBE	OEDEMATOUS
129	21/16	SARAVANAN	45	MALE	DRIVER	PEDESTRAN VS TWO WHEELER	7.45 PM	PEDESTRAN	YES	9 DAYS	NO	RT FTP	NO	NO	NO	NO	INTACT	RT FTP	INTACT	NO	FRACTURE RIBS	NO	NORMAL
130	22/16	SUDHAGAR	25	MALE	PAINTER	PEDESTRAN VS TWO WHEELER	11.00 AM	PEDESTRAN	YES	4 DAYS	NO	LT FTP	NO	LT FTP	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	LT FTP	INTACT	NO	NO	NO	OEDEMATOUS
131	23/16	SATHYA	12	FEMALE	PLUMBER	PEDESTRAN VS TWO WHEELER	1.30 PM	PEDESTRAN	YES	2 DAYS	NO	DIFFUSE	NO	NO	NO	PRESENT	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
132	24/16	THIAGARAJAN	20	MALE	COOK	TWO WHEELER VS TWO WHEELER	3.00 PM	TWO WHEELER DRIVER	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
133	25/16	SAROJA	45	MALE	TEACHER	TWO WHEELER VS FOUR WHEELER	11.00 AM	TWO WHEELER DRIVER	YES	3 DAYS	NO	RT FTP	NO	NO	NO	NO	FISSURE FRACTURE RT PARIETAL BONE AND TEMPORAL BONE	DIFFUSE	FF RT MCF	NO	NO	LACERATION RT FRONTAL LOBE	OEDEMATOUS
134	27/16	DINESH	20	MALE	COOLIE	SELF FALL FROM TWO WHEELR	9.30 PM	TWO WHEELER PILLION RIDER	YES	7 DAYS	NO	RT FTP	NO	RT FTP	PRESENT	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
135	28/16	UNKNOWN	40	MALE	COOLIE	PEDESTRAN VS TWO WHEELER	12.45 PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
136	234/16	SUBRAMANI	45	MALE	DRIVER	TWO WHEELER VS TWO WHEELER	8.00 PM	TWO WHEELAR DRIVER	YES	4 DAYS	NO	RT FTP	NO	NO	NO	NO	COMMINUTED FRACTURE RT FRONTAL BONE	RT FTP	COMMINUTED FRACTURE RT ACF	NO	NO	NO	OEDEMATOUS
137	235/16	KOTHANDARAMAN	74	MALE	COOLIE	PEDESTRAN VS TWO WHEELER	12.30 PM	PEDESTRAN	YES	1 DAY	NO	LT FTP	NO	NO	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
138	243/16	RAMAN	60	MALE	retired	PEDESTRAN VS TWO WHEELER	4.35 PM	PEDESTRAN	YES	14 DAYS	YES	DIFFUSE	LT PARIETAL	DIFFUSE	NO	NO	BONY AND DURAL DEFECT	LT FTP	INTACT	NO	CHEST INJURY	NO	OEDEMATOUS
139	258/16	BABU	39	MALE	SELF EMPLOYED	TWO WHEELER VS TWO WHEELER	12.30 PM	WHEELER DRIVER	YES	7 DAYS	NO	NO	NO	RT FTP	NO	PRESENT	FISSURE FRACTURE RT PARIETAL AND TEMPORAL BONE	RT FTP	INTACT	NO	NO	NO	NORMAL
140	2101/16	MOHAMMAD ISMAIL	54	MALE	COOLIE	SELF FALL FROM TWO WHEELR	11.00 AM	TWO WHEELER DRIVER	YES	2 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
141	2104/16	DEEPAK	6	MALE	STUDENT	PEDESTRAN VS TWO WHEELER	6.00 PM	PEDESTRAN	YES	4 DAYS	NO	RT FTP	NO	RT FTP	PRESENT	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
142	2105/16	PRATAPAN	52	MALE	COOLIE	PEDESTRAN VS HEAVY VEHICLE	3.20 PM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
143	2107/16	GOVINDAMMAL	55	MALE	PAINTER	SELF FALL FROM TWO WHEELR	6.20 PM	TWO WHEELER PILLION RIDER	YES	13 DAYS	NO	NO	NO	RT FTP	NO	NO	FISSURE FRACTURE RT PARIETAL AND TEMPORAL BONE	RT FTP	INTACT	NO	NO	NO	NORMAL
144	2108/16	FEROZ	28	MALE	SELF EMPLOYED	TWO WHEELER DRIVER	2.00 PM	TWO WHEELER DRIVER	YES	1 DAY	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT PARIETAL AND TEMPORAL BONE	RT FTP	COMMINUTED FRACTURE	NO	NO	NO	NORMAL
145	2109/16	SUNDERESAN	75	MALE	retired	PEDESTRAN VS CAR	11.00 PM	PEDESTRAN	YES	9 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	FF RT MCF	NO	NO	NO	NORMAL
146	2579/15	PARTHIBAN	45	MALE	DRIVER	PEDESTRAN VS CAR	11.00 PM	PEDESTRAN	YES	5 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	INTACT	NO	NO	NO	NORMAL
147	2255/16	VELMURUGAN	44	MALE	COOLIE	SELF FALL FROM TWO WHEELR	3.00 PM	TWO WHEELER DRIVER	YES	1 DAY	YES	LT FTP	LT PARIETAL	LT FTP	NO	NO	BONY AND DURAL DEFECT LEFT FTP	LT FTP	INTACT	NO	NO	NO	OEDEMATOUS
148	1937/16	MANGAMMAL	64	FEMALE	HOUSE WIFE	PEDESTRAN VS CAR	3.25 PM	PEDESTRAN	YES	9 DAYS	NO	RT FTP	NO	RT FTP	NO	PRESENT	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
149	2140/15	PRAKASH	32	MALE	COOLIE	SELF FALL FROM TWO WHEELR	2.00 PM	TWO WHEELER DRIVER	YES	1 DAY	YES	LT FTP	MID PARIETAL	LT FTP	NO	NO	BONY AND DURAL DEFECT LT FTP	RT FTP	INTACT	NO	NO	LACERATION LT TEMPORAL LOBE	OEDEMATOUS

150	900/16	MURUGAN	45	MALE	SELF EMPLOYED	PEDESTRAN HIT BY UNKNOWN VEHICLE	5.00 AM	PEDESTRAN	YES	12 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	RT FTP	LT PCF	NO	NO	LACERATION LT TEMPORAL LOBE	OEDEMATOUS
151	1468/16	SURYA PRAKASH	19	MALE	STUDENT	PEDESTRAN VS TWO WHEELER	2.00 PM	PEDESTRAN	YES	6 DAYS	YES	LT FTP	NO	DIFFUSE	NO	NO	BONY AND DURAL DEFECT LT FTP	LT FTP AND OCCIPITAL	RT PCF	NO	LIVER LACERATION	LACERATION LT TEMPORAL LOBE	OEDEMATOUS
152	1558/16	VENKETESH RAO	40	MALE	DEFENCE	TWO WHEELER VS FOUR WHEELER	3.00 PM	CYCLIST	YES	3 DAYS	YES	DIFFUSE	NO	DIFFUSE	NO	NO	BONY AND DURAL DEFECT LT FTP	DIFFUSE	CENTRE OF PCF	NO	NO	NO	NORMAL
153	2141/16	SEETHA	55	FEMALE	HOUSE WIFE	PEDESTAN VS TWO WHEELER	5.00 PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NO	INTACT	RT FTP	INTACT	NO	CHEST INJURY	NO	NORMAL
154	772/16	SANOJ	22	MALE	STUDENT	SELF FALL FROM TWO WHEELR	2.00 PM	TWO WHEELER RIDER	BROUGHT DEAD		NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	LT FTP AND OCCIPITAL	LT PCF	NO	NO	NO	NORMAL
155	623/17	DHANAPAL	35	MALE	COOLIE	TWO WHEELER VS FOUR WHEELER	3.00 PM	TWO WHEELER DRIVER	YES	9 DAYS	NO	DIFFUSE	NO	DIFFUSE	PRESENT	NO	DEPRESSED FRACTURE LT PARIETAL BONE	LT FTP	INTACT	NO	NO	NO	OEDEMATOUS
156	2101/16	SADHAN	25	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELER	11.00 PM	TWO WHEELER DRIVER	YES	2 DAYS	NO	LT FTP	NO	LT FTP	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	DIFFUSE	LT MCF	NO	RIB FRACTURE	NO	NORMAL
157	2102/16	BABY	39	FEMALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	6.00 PM	PEDESTRAN	YES	4 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	INTACT	NO	INJURY TO RT LOWER LIMB	NO	NORMAL
158	2105/16	RAKKAMMAL	74	MALE	HOUSE WIFE	PEDESTRAN VS TWO WHEELER	3.20 PM	PEDESTRAN	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	RIB FRACTURE	NO	NORMAL
159	2106/16	DEV	64	MALE	COOLIE	SELF FALL FROM TWO WHEELR	6.20 PM	TWO WHEELER DRIVER	YES	13 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	INTACT	RT FTP	RT MCF	NO	NO	NO	NORMAL
160	2108/16	KANNIYAPPAN	47	MALE	DRIVER	PEDESTRAN VS CAR	2.00 PM	PEDESTRANLT	YES	1 DAY	NO	RT FTP	NO	RT FTP	NO	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
161	174/16	unKNOWN	50	MALE	COOLIE	PEDESTRAN VS	12.3O PM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT FRONTAL BONE AND PARIETAL BONE	DIFFUSE	INTACT	NO	CHEST INJURY	NO	NORMAL
162	177/16	ANJALAI	60	FEMALE	HOUSEWIFE	PEDESTRN VS FOUR WHEELER	7,30 PM	PEDESTRAN	YES	3 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT PARIETAL BONE	DIFFUSE	RT MCF	NO	NO	NO	NORMAL
163	179/16	SELVI	44	FEMALE	HOUSEWIFE	PEDESTRN VS FOUR WHEELER	9.00 PM	PEDESTRAN	YES	3 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT PARIETAL AND TEMPORAL BONE	DIFFUSE	INTACT	NO	NO	NO	NORMAL
164	184/16	RAJESH	21	MALE	COOLIE	SELF FALL FROM TWO WHEELR	7.00 PM	TWO WHEELER DRIVER	YES	6 DAYS	NO	LT FTP	NO	LT FTP	NO	NO	FISSURE FRACTURE LT TEMPORAL BONE	LT FTP	LT MCF	NO	NO	NO	NORMAL
165	185/16	UNKNOWN	50	MALE	DRIVER	SELF FALL FROM TWO WHEELR	9.00 PM	TWO WHEELER DRIVER	YES	6 DAYS	NO	DIFFUSE	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
166	188/16	INNAT ALI	32	MALE	COOLIE	TWO WHEELER VS CAR	4.00 PM	TWO WHEELER DRIVER	YES	5 DAYS	NO	LT FTP	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
167	192/16	MUNUSWAMY	65	MALE	SELF EMPLOYED	TWO WHEELER VS CAR	5.30 PM	TWO WHEELER DRIVER	YES	6 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
168	193/16	SASIKUMAR	39	MALE	SELF EMPLOYED	TWO WHEELR VS FOUR WHEELER	2.00 PM	TWO WHEELER DRIVER	YES	3 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	NO	NO	NORMAL
169	194/16	SELVARAJ	64	MALE	COOLIE	SELF FALL FROM TWO WHEELER	8.30 PM	PILLION RIDER	YES	1 DAY	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT PARIETAL BONE AND TEMPORAL	LT FTP	LT MCF	NO	NO	NO	OEDEMATOUS
170	197/16	MANIKAM	22	MALE	COOLIE	SELF FALL FROM TWO WHEELER	3.45 PM	PILLION RIDER	YES	5 DAYS	NO	RT FTP	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
171	2120/16	CHAKRAVATHI	64	MALE	COOLIE	SELF FALL FROM TWO WHEELER	3.30 PM	TWO WHEELER PILLION RIDER	YES	1 DAY	NO	NO	NO	LT FTP	NO	NO	FISSURE FRACTURE LT PARIETAL BONE	DIFFUSE	LT MCF	NO	NO	NO	OEDEMATOUS
172	2121/16	SURYA PRAKASH	34	MALE	SELF EMPLOYED	SELF FALL FROM TWO WHEELER	6.20 PM	TWO WHEELER DRIVER	YES	2 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	FISSURE FRACTURE RT PARIETAL BONE AND TEMPORAL	DIFFUSE	INTACT	NO	NO	NO	NORMAL
173	2122/16	VENKATESH	26	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	2.00 PM	CAR PEDESTRAN	YES	2 DAYS	NO	LT FTP	NO	LT FTP	PRESENT	NO	INTACT	LT FTP	INTACT	NO	NO	NO	NORMAL
174	2124/16	GANESAN	18	MALE	DRIVER	SELF FALL FROM TWO WHEELER	11.00AM	TWO WHEELER PILLION RIDER	YES	5 DAYS	NO	DIFFUSE	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
175	2128/16	ANSARI	23	MALE	PAINTER	SELF FALL FROM TWO WHEELER	4.00 AM	TWO WHEELER DRIVER	YES	2DAYS	NO	DIFFUSE	NO	NO	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
176	89/16	RAMACHANDRAN	65	MALE	COOLIE	SELF FALL FROM TWO WHEELER	9.00 PM	TWO WHEELER DRIVER	YES	9 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	LT FTP	INTACT	NO	NO	NO	OEDEMATOUS
177	90/16	JAYA	31	FEMALE	HOUSE WIFE	SELF FALL FROM TWO WHEELER	7.30PM	TWO WHEELER DRIVER	YES	7 DAYS	NO	LT FTP	NO	LT FTP	NO	NIL	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
178	91/16	SUNDARI	70	FEMALE	HOUSE WIFE	SELF FALL FROM BUS	3.00 PM	PASSENGER	BROUGHT DEAD		NO	DIFFUSE	RT PARIETAL	DIFFUSE	NO	PRESENT	COMMINUTED FRACTURE VAULT OF SKULL	DIFFUSE	MCF	NO	NO	NO	OEDEMATOUS
179	93/16	RAMU	30	MALE	COOK	SELF FALL FROM TWO WHEELER	1.00 PM	TWO WHEELER RIDER	YES	2 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	FISSURE FRACTURE LT PARIETAL BONE	LT FTP	INTACT	NO	NO	NO	NORMAL
180	94/16	SANKARI	52	FEMALE	HOUSEWIFE	PEDESTRAN VS HEAVY VEHICLE	2.00 PM	PEDESTRAN	YES	2 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	INTACT	NO	NO	NO	NORMAL
181	851/16	THARINI	41	FEMALE	HOUSEWIFE	SELF FALL FROM TWO WHELER	7.00AM	TWO WHEELAR DRIVER	BROUGHT DEAD		NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	LT FTP	RT ACF	NO	NO	NO	NORMAL
182	309/16	SHANKAR	53	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	8.00 PM	PEDESTRAN	YES	8 DAYS	YES	NO	RT PARIETAL	NO	NO	NO	BONY AND DURAL DEFECT RT FTP	RT FTP	COMMINUTED FRACTURE RT ACF AND LT ACF	NO	NO	LACERATION BOTH FRONTAL	OEDEMATOUS
183	376/16	ELLAMMAL	60	FEMALE	HOUSEWIFE	PEDESTRAN VS TWO WHEELER	4.00 PM	PEDESTRAN	YES	9 DAYS	YES	DIFFUSE	RT PARIETAL	DIFFUSE	NO	NO	BONY AND DURAL DEFECT LT FTP	LT FTP AND OCCIPITAL	RT PCF	NO	NO	LACERATION RT CEREBELLUM	OEDEMATOUS
185	2434/16	ARUMUGAM	30	MALE	COOLIE	TWO WHEELER VS TWO WHEELER	5.00 PM	TWO WHEELER DRIVER	YES	5 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	INTACT	NO	NO	NO	NORMAL
186	187/16	RAJESH	21	MALE	STUDENT	TWO WHEELER VS TWO WHEELER	12.00PM	TWO WHEELAR DRIVER	YES	4 DAYS	NO	DIFFUSE	NO	DIFFUSE	NO	NO	INTACT	DIFFUSE	RT PCF	NO	NO	NO	OEDEMATOUS
187	2521/16	MAHESWARI	21	FEMALE	STUDENT	SELF FALL FROM TWO WHEELER	8.00 PM	TWO WHEELER PILLION RIDER	BROUGHT DEAD		NO	NO	NO	DIFFUSE	NO	PRESENT	INTACT	RT FTP	INTACT	NO	NO	NO	OEDEMATOUS
188	2051/16	UNKNOWN	35	MALE	NK	PEDESTRAN VS TWO WHEELER	10.00PM	PEDESTRAN	YES	8 DAYS	NO	LT FTP	NO	NO	NO	NO	INTACT	LT FTP	INTACT	NO	INJURY TO RT LOWER LIMB	NO	NORMAL
189	190/16	UNKNOWN	70	MALE	NK	PEDESTRAN VS HEAVY VEHICLE	2.00 PM	PEDESTRAN	YES	2 DAYS	NO	RT FTP	NO	RT FTP	NO	PRESENT	FISSURE FRACTORE RT TEMPORAL BONE	RT FTP	RT ACF AND RT MCF	NO	INJURY TO RT UPPER LIMB	NO	NORMAL
190	167/16	GOVINDAN	52	MALE	COOLIE	PEDESTRAN VS HEAVY VEHICLE	3.00 PM	PEDESTRAN	BROUGHT DEAD		NO	DIFFUSE	NO	DIFFUSE	NO	PRESENT	COMMINUTED FRACTURE OF VAULT OF SKULL	DIFFUSE	COMMINUTED FRACTURE BASE OF SKULL	NO	CHEST INJURY	BIFRONTAL CONTUSION	OEDEMATOUS
191	1981/16	CHANDRA BORA	29	MALE	DAILY WAGE	TWO WHEELER VS TWO WHEELER	5.00 PM	TWO WHEELER RIDER	YES	2 DAYS	NO	DIFFUSE	LT PARIETAL	DIFFUSE	NO	NO	INTACT	DIFFUSE	HINGE FRACTURE RT MCF TO LT MCF	NO	NO	NO	NORMAL
192	1935/16	BAHADUR SINGH	31	MALE	CONSTUCTION WORKER	TWO WHEELER VS LORRY	1.15 PM	TWO WHEELER DRIVER	YES	1 DAY	NO	DIFFUSE	NO	NO	NO	NO	INTACT	RT FTP	INTACT	NO	NO	NO	NORMAL
193	1973/16	ARUN	26	MALE	TAILOR	PEDESTRAN VS FOUR WHEELER	3.30 PM	PEDESTRAN	YES	3 DAYS	NO	DIFFUSE	NO	NO	NO	NO	INTACT	RT FTP	RT PCF	NO	NO	LACERATION LT TEMPORAL	OEDEMATOUS
194	1896/16	KARUPPAN	74	MALE	retired	PEDESTRAN VS TWO WHEELER	4.25 PM	PEDESTRAN	YES	1 DAY	NO	NO	NO	DIFFUSE	NO	PRESENT	INTACT	DIFFUSE	INTACT	NO	CHEST INJURY	NO	NORMAL
195	1899/16	SAMEER BASHA	16	MALE	STUDENT	CYCLE VS FOUR WHEELER	3.45 PM	CYCLIST	YES	6 HOURS	NO	DIFFUSE	NO	NO	NO	NO	DEPRESSED FRACTURE CORONAL SUTURE	DIFFUSE	RT PCF	NO	NO	NO	NORMAL
196	1944/16	MADHAVAN	35	MALE	COOLIE	TWO WHEELER VS FOUR WHEELER	1.25 PM	MOTORCYCLIST	YES	2 DAYS	YES	DIFFUSE	RT PARIETAL	DIFFUSE	NO	NO	BONY AND DURAL DEFECT RT FTP	DIFFUSE	INTACT	NO	NO	NO	NORMAL
197	1940/16	KUMAR	64	MALE	TAILOR	PEDESTRAN VS TWO WHEELER	9.10 PM	PEDESTRAN	YES	8 HOURS	YES	DIFFUSE	RT PARIETAL	DIFFUSE	NO	NO	BONY AND DURAL DEFECT RT FTP	DIFFUSE	INTACT	NO	NO	NO	OEDEMATOUS
198	465/16	DILLI	59	MALE	SELF EMPLOYED	PEDESTRAN VS TWO WHEELER	9.15 PM	PEDESTRAN	YES	20 H0URS	NO	NO	NO	DIFFUSE	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	DIFFUSE	RT MCF	NO	NO	NO	OEDEMATOUS
199	902/16	MAHENDRAN	43	MALE	COOLIE	PEDESTRN VS FOUR WHEELER	2.00 AM	PEDESTRAN	YES	1 DAY	NO	DIFFUSE	NO	NO	NO	NO	FISSURE FRACTURE OCCIPITAL BONE	RT FTP	RT PCF	NO	NO	LACERATION RT FRONTAL LOBE	OEDEMATOUS
200	904/16	VENKATARATHINAM	36	MALE	COOLIE	SELF FALL FROM TWO WHEELER	6.00 PM	TWO WHEELER DRIVER	YES	6 DAYS	NO	RT FTP	NO	RT FTP	NO	NO	FISSURE FRACTURE RT TEMPORAL BONE	RT FTP	RT MCF	NO	NO	NO	NORMAL